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APPOINTMENTS.

No charge is made for the insertion of these notices; the necessary details should be sent before the 18th of each month to The Editor, THE PRACTITIONER, Howard Street, Strand, London, W.C. 2, to secure inclusion.

CHADWICK, M., B.A. Camb., M.R.C.S., L.R.C.P. Lond., appointed Medical Officer of the King's Lynn Union Institution.

GABRIEL, V., F.R.C.S.Eng., appointed Medical Superintendent, Shoreditch Parish Infirmary.

OLDFIELD, CARLTON, M.D., B.S. Lond., F.R.C.S., appointed Obstetric Surgeon to the Leeds Infirmary.

MUNBY, W. MAXWELL, M.B., Ch.B., F.R.C.S., appointed Aural Surgeon to the Leeds Infirmary.

COLLINSON, HAROLD, C.M.G., D.S.O., M.S., F.R.C.S., appointed Surgeon in Charge of Out-patients to the Leeds Infirmary.

STEWART, A. G., Lieut.-Colonel, appointed to the command of Paddington Military Hospital.

STEWART, MATTHEW J., M.B., M.R.C.P., appointed Pathologist to the Leeds Infirmary.

TOWNSEND, ARTHUR, M.D., appointed Medical Superintendent of Barnwood House, Gloucester, in place of Dr. James Greig Soutar, resigned.

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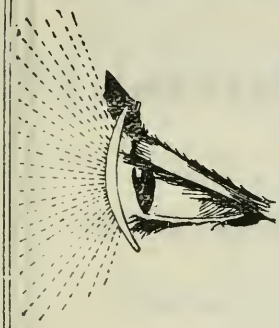

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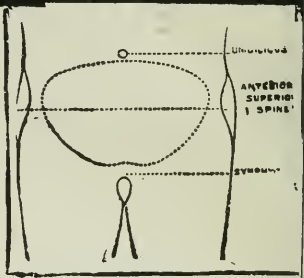

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MEDICAL PRESS · 1913 II 321,322,323 1918. II 283.
JOURNAL OF THE ROYAL NAVAL MEDICAL SERVICE: OCTOBER 1918
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A pluriglandular preparation with Spermin as its base and put up in soluble gelatine capsules. Widely prescribed, with exceptionally gratifying results, in Nervous and Mental Instability, Neuroses, Psychoses, Shock, Insomnia, and in conditions resulting from glandular insufficiency, metabolic disorder, imperfect oxidation, impairment of nutrition, Fatigue and exhaustion, and Impotentia.

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> "Dr. ——, M.D., M.B., writes to say that he has treated many patients with trimethenal allylic carbide and that he can endorse the results recorded by Mr. ——, M.R.C.S., in the "Lancet" of November 9th. Five patients constantly using preparations of allyl for other ailments, and residing in different houses in which the inmates were all stricken down with influenza, have themselves remained immune."

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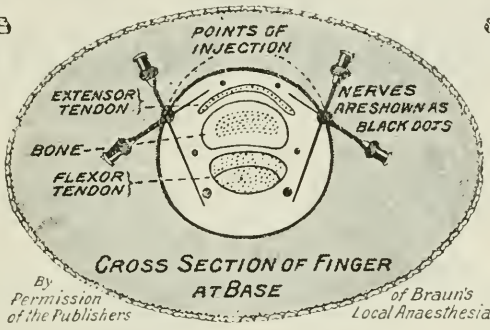

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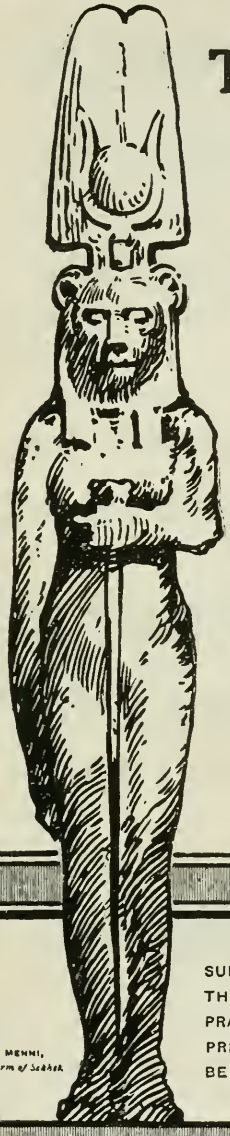

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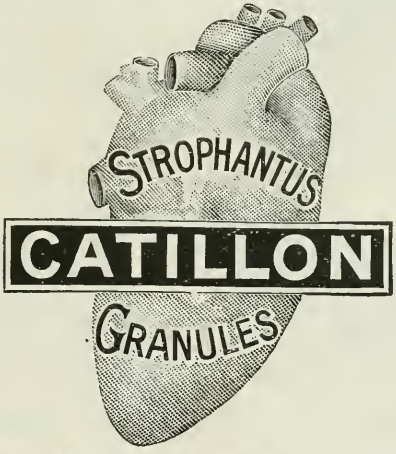

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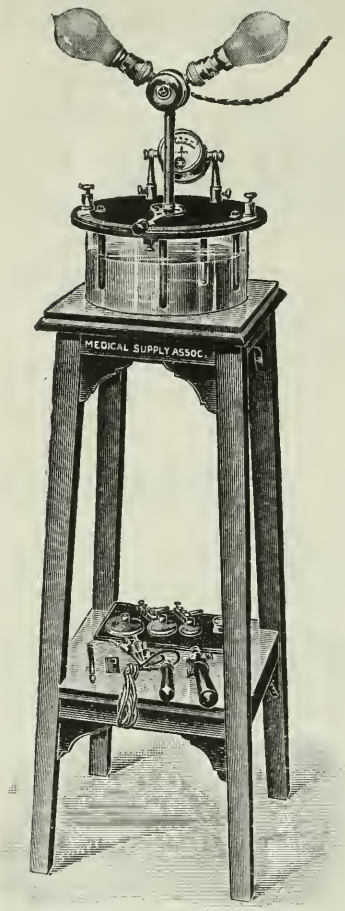

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Antiseptic, Disinfectant, Germicide, Deodorant.

Internal, Powder, External.

THE PRACTITIONER.

FEBRUARY, 1919.

Special Influenza Number.—Part II.

INFLUENZA AS AFFECTING THE RESPIRATORY SYSTEM.

By HECTOR MACKENZIE, M.A., M.D., F.R.C.P.

Physician to St. Thomas's Hospital; Consulting Physician to the Brompton Hospital for Consumption and Diseases of the Chest; Consulting Physician to King Edward VII. Sanatorium, Midhurst

At the present day it seems doubtful whether the specific cause of epidemic influenza has yet been discovered, and it is prudent to consider it as belonging to the group of infectious diseases of doubtful or unknown ætiology, which includes all the most typical contagious diseases, like small-pox, measles, typhus, scarlet fever, etc. It seems likely that the true virus is some ultra-microscopic organism, which later on will be revealed to us. The reasons why Pfeiffer's bacillus, the so-called influenza bacillus, is not unreservedly accepted as the causal organism are elsewhere discussed. Since Pfeiffer isolated and cultivated it in 1892, it has been found with more or less constancy in the secretions from the respiratory tract, and in the lesions of the respiratory system, and there can be no doubt but that it plays a very large part in the production of the respiratory complications of influenza which we are now to consider; but it is not the only microbe which seems to follow in the train of the unknown virus, for it is commonly associated with pneumococci, streptococci, etc. The virus of influenza is like the unclean spirit mentioned in the Bible, which "taketh with him seven other spirits more wicked than himself and they enter in and dwell there; and the last state of that man is worse than the first."

The respiratory system seems to be the favourite battle-ground selected for the malign influence and its accomplices, the influenza bacillus, the pneumococcus, and the various strains of streptococci. It would seem that at the beginning of an epidemic pulmonary complications are not so common as later on; moreover, attempts to isolate the influenza bacillus are seldom successful at this stage; but later respiratory complications are more common, and the presence of the B. influenzæ becomes the rule instead of the exception. Respiratory troubles are the most common features of the disease, as it has been observed from the Middle Ages. In the earliest accounts we have, those

of the 12th century, special reference is made to cough as a chief symptom, and from its intensity it was spoken of as " tussis magna," " tussis intolerabilis et inaudita," or " tussis gravissima." The cough " disturbed the patients night and day," and in somewhat later accounts was described as " troublesome and attended with copious expectoration," or tormenting or distressing, and often as accompanied with dyspnœa sometimes severe. Hence such names as the following were given to the malady:—Tussis popularis, febris suffocativa, gravedo anhelosa, der Schafhusten.

Thomas Short, in his *Chronological History of the Air, Weather, etc.*, says that in 1548 all Europe was sore afflicted with a pestilential peripneumony, with blood-spitting and difficulty of breathing. In 1557, there was a run of defluctions, coughs, fevers, pain in the side, asthma, many fatal peripneumonies, and in 1563 cough, pleurisies, and peripneumonies were prevalent. At the present day we cannot say with certainty that these were cases of influenza, but the symptoms are very like what we have been seeing during the present epidemic of influenza.

During the epidemics of the nineties, and of the first two decades of this century, a distressing, hard, frequent cough has been one of the most common symptoms of the disease, as it was in former times, and we have only too often seen the serious complications of bronchitis, broncho-pneumonia, pneumonia, and pleurisy coming on during the height of the disease or while it ought to be subsiding. A considerable number of cases of influenza have been followed by the development of pulmonary tuberculosis.

I will first describe the morbid changes which are met with in the respiratory tract, whether as part of the disease or as complications.

The larynx is frequently affected, but seldom severely so. The vocal cords may be congested and swollen, and sometimes are intensely red. In rarer cases, sometimes with and sometimes without involvement of the cords, there is swelling and congestion of the ventricular bands, the subglottic tissue, and the arytenoid folds.

The mucous membrane of the trachea is specially liable to attack. It is diffusely reddened, and is coated with thick viscid mucus.

The mucous membrane of the bronchi shows similar changes. The ciliated epithelium is partly destroyed, detached fragments are shed into the interior of the tubes, and some of the cells are separated from their base by leucocytes. Leucocytes also line the free surface and lie in small groups on apparently intact epithelial cells, or they may penetrate between the cells or fill the spaces left by the cells which have been shed. The capillaries and venules in the bronchial wall are distended with blood, and the peribronchial tissue is invaded by migratory cells. The affection of the trachea and bronchi has been compared with, and appears to be analagous to, that of the urethra and conjunctiva set up by the gonococcus.

In the secretion from the trachea and bronchi, Pfeiffer's bacillus is frequently found to be present, sometimes in almost pure culture.

This organism is also often present in great numbers lying over the epithelial cells as well as between them. Clumps of bacilli may be found in the epithelial cells themselves or in the interior of the leucocytes which make their way between them, but in the submucous tissue the bacilli may be met with singly.

The lumen of some of the smaller bronchi is more or less filled with finely granular material consisting of coagulated mucus with numerous leucocytes embedded in it. The alveoli may be similarly plugged, and they contain not only leucocytes, but also numerous swollen cells which are often pigmented and are regarded by Pfeiffer as altered epithelial cells.

The most common morbid change in the lungs is broncho-pneumonia. It was present in 54 out of 76 fatal cases examined during the recent epidemic in the British Armies in France. Numerous scattered areas of lobular consolidation may be seen, affecting an extensive tract but separated by small air-containing patches. The cut surface is somewhat mottled and smooth or slightly granular The consolidation is due rather to a cellular inflammation than to a fibrinous exudation. The solid areas more resemble spleen than liver substance, and the lung may be said to be splenicized instead of hepatized.

Pfeiffer described small patches of consolidation, from the size of a pin's head to that of a pea, separated by areas of a greyish-yellow colour from the surrounding dark red tissue. At the centres of the consolidated areas, yellowish-grey, thick and very tenacious pus may be seen exuding from the cut sections of the bronchi. Not only Pfeiffer's bacilli, but pneumococci, streptococci, or staphylococci may be present in the consolidated islets, some within, some outside, the leucocytes and the epithelial cells. In some cases, and this has again been observed in the epidemic in France, the pneumonic areas are small and shotty, closely resembling miliary tubercles. The microscope showed these to consist of consolidated lung with fibrinous exudate surrounding inflamed bronchioles. This form has been appropriately called " miliary pneumonia."

At a later stage small abscesses may form. Some lobular areas are entirely filled with leucocytes and, merging into one another, form small centres of suppuration.

Subpleural and interstitial hæmorrhages are observed in some cases. These, however, are usually small and localized.

Pneumonic consolidation of the lobar type is also met with, but is much less common than the broncho-pneumonic form. The appearances of the consolidated lung resemble those of ordinary pneumonia. The upper lobes seem to be more often affected than in primary pneumonia. The pneumococcus or the streptococcus pyogenes may be found to be present.

Influenzal pneumonia is sometimes slow to undergo resolution. The affected areas may be transformed into cicatricial connective tissue, and be replaced by firm greyish-red fibrous masses of almost cartilaginous consistence. Gangrene of the lung sometimes

occurs.

The pleuræ are frequently affected. Flakes of fibrinous exudation, patchy or diffuse, varying in thickness as well as in size, may be observed on the surface of the lungs. These consist chiefly of masses of leucocytes containing crowds of micro-organisms.

When there is effusion, it is more commonly sero-purulent than purulent. The effusion is seldom a large one, and commonly does not exceed a few ounces. It has a peculiar, cloudy, rather yellow appearance, and has been likened to clay water or a suspension of cream of tartar.

SYMPTOMS.

The affection of the larynx, although seldom of much intensity, usually results in some huskiness or weakness of the voice, hoarseness, or aphonia. Soreness in the region of the larynx, with some pain on swallowing, is not uncommon. These symptoms are more decided, if laryngitis is severe.

The presence of tracheitis gives rise to distinctive features. The diffuse congestion of the mucous membrane results in pain along the course of the trachea, and a hacking and persistent cough, sometimes very severe, or paroxysmal like whooping cough, occurring night and day, and causing much distress to the patient. The cough is often at first dry, or attended with very scanty glutinous expectoration dislodged with difficulty. The cough frequently continues after the acute stage has long passed away. It may be so severe as to lead to rupture of some of the fibres of the abdominal muscles, as in a case which I saw in consultation. The patient during a fit of coughing was seized with pain in the abdomen, and shortly after a tumour the size of a cricket ball was observed in the left hypochondrium. The presence of this tumour gave rise to a suspicion of carcinoma, but I diagnosed a hæmatoma, and the swelling gradually disappeared in the course of two or three months.

When the bronchi are first affected, there is merely hyperæmia, and hardly any sign of secretion. Swelling of the mucous membrane often ensues, which is manifested by a louder respiratory murmur at certain points, or by a harsher inspiratory sound. The breathing becomes shorter and more rapid, and at times there is actual dyspnœa. The presence of scattered patches of atelectasis may give rise to slight dulness on percussion, and diminished respiratory murmur. This, the stage of dry catarrh, is usually of short duration, and before long there is secretion from the mucous membrane, and, on auscultation, a great variety of râles and rhonchi will be heard; when the smaller tubes are affected, characteristic râles are audible at the bases, the sharp high-pitched "sticky" râles of Goodhart, to which I shall have occasion to refer later. The sputum, which is usually purulent from the first, is commonly yellow, or greenish-yellow, but is sometimes tinged with blood or has a peculiar flesh-coloured tint. It is sometimes nummular, sometimes viscid, and sometimes almost

diffluent. In some cases it is profuse, and may amount to one or two pints a day. Occasionally it contains fibrinous casts.

Tracheitis and bronchitis may be regarded rather as integral parts of the disease when they are slight, but broncho-pneumonia is a true complication, and when it occurs is always serious. It frequently supervenes on bronchitis, and it is difficult to draw any sharp clinical distinction between severe bronchitis and broncho-pneumonia, which are generally co-existent. The symptoms, therefore, of severe bronchitis and broncho-pneumonia may be described together. The patient soon is obviously very ill. The pulse and respiration are rapid. The temperature often runs high and is irregular, following no definite type. The face is flushed or congested, or there is obvious cyanosis. The breathing is distressed, and the patient lies propped up with pillows battling for breath. Air-hunger is often a prominent symptom. Cyanosis and prostration rapidly increase when the illness is approaching a fatal termination. In elderly subjects, the height of the temperature is no guide to the severity of the illness. The sputum resembles that already described as characteristic of bronchitis. In a good many of the cases I have seen during the present epidemic, the sputum has contained blood, but rusty coloured sputum has been rare. The physical signs are usually most in evidence at the bases. There a certain degree of dulness may be made out, and fine and coarse bubbling râles are present with sometimes distinct but distant bronchial breathing. In some cases there are similar signs at one or other apex.

The signs are apt to wander irregularly. We may find that first the lower lobe of one lung is affected, and then the upper lobe of the other. Patches of dulness are not sharply defined, but gradually merge in resonant parts, and the dulness hardly ever is so pronounced as in lobar pneumonia. The borders of the lungs are likely seats for these patches. The presence of bronchial breathing is by no means constant, and, on account of the blocking of bronchi leading to the affected areas either by swelling of the mucous membrane or by profuseness of secretion, the breath sounds are often very feeble or almost inaudible. When the breath sounds are bronchial, they are nearly always distant, and the tubular breathing typical of lobar pneumonia is very exceptional.

The râles audible at the bases of the lungs, already mentioned as the " sticky " râles of Goodhart, are very characteristic. They have been called by Wintrich " vesicular," and they give one the impression of the bursting of a large number of air bubbles of uniform size. Wintrich suggests that they are due to the sudden tearing apart during inspiration of the walls of the smaller bronchi and alveoli which have been stuck together by mucus. This might conceivably occur when there is swelling of the mucous membrane as well as a lining of tenacious mucus. If this explanation is correct, and it seems to be supported by the fact that even forcible coughing and expectoration do not alter the character of the sounds, the term " sticky " is not inappropriate, although not altogether a suitable

adjective apart from this to apply to a sound.

An unfavourable termination to broncho-pneumonia or capillary bronchitis is, unfortunately, frequent. The dyspnœa increases, cyanosis deepens, the pulse becomes more feeble and more rapid, delirium comes on, prostration increases, and the patient gradually sinks. In patients who recover, improvement sets in slowly, the fever gradually subsides, and the lung takes time to clear up. Relapses are not uncommon.

While, as a rule, broncho-pneumonia is associated with bronchitis, it may occur independently, in which case there may be no expectoration throughout or until the advent of resolution. One finds the physical signs of consolidated areas in the lung with abundant fine râles such as one gets in the early stage of lobar pneumonia, but with no moist sounds and rhonchi.

When the pneumonia is of the lobar form, it is usually pneumococcic or streptococcic in origin. The constitutional symptoms are out of proportion to the physical signs. Pain in the side and a pleural rub is common at the beginning. The cough may be dry at first, but later the sputum is muco-purulent and often is bloodstained or sanguineous. Dyspnœa is usually considerable. Signs of consolidation may be delayed, but later become well-marked and extend over a large area, affecting both lower lobes, one apex, or the whole of one lung. The temperature is usually high but irregular, and falls more frequently by lysis than by crisis. Profuse sweats are common without a corresponding fall in the temperature. Prostration, delirium, sleeplessness, restlessness, rapid pulse and respiration, and cyanosis supervene in the graver cases. But while there is life there is hope, and recovery has followed in cases which seemed desperate.

It has been estimated that pneumonia, or broncho-pneumonia, is met with as a complication of influenza in from 5 to 7 per cent. of the cases, but it is probable that this estimate is too high. Only 1 per cent. of all cases of influenza in the First British Army, 1918, developed pneumonia or broncho-pneumonia. I have seen many cases of these complications, and, if I may judge from my own experience, an unduly large number of members of our own profession and of nurses have been attacked. Few doctors could say with the cheery practitioner of 1833 : " Best thing I ever had. Quite a godsend. Everybody ill, nobody dying." A physician sees only the severe cases, both in his hospital and in his private practice; any conclusions he can form, as to the actual frequency of serious respiratory complications among a population attacked with influenza, must be fallacious. The danger to life of influenzal broncho-pneumonia and pneumonia certainly appears to be grave, and from 17 to 43·9 per cent. of the cases prove fatal. Even in a select body of men like the first British Army, 10 to 15 per cent. of the cases of pneumonia terminated fatally.

Pleurisy co-exists in from 12 to 33 per cent. of the cases with bronchitis, broncho-pneumonia, or pneumonia. As a general rule, it is sero-fibrinous, but it is sometimes purulent. In influenzal empyemata, the streptococcus pyogenes has been found to be present. A form of

acute primary pleurisy has been described, which comes on nearly simultaneously with the onset of influenza. This is generally an extremely grave affection, and is characterized by chills, high fever, severe dyspnœa, and cyanosis.

The complications which have hitherto been described have been of an acute nature, but chronic affections, chronic bronchitis, and chronic pneumonia may also be set up, and left behind after an attack of influenza. Cough and expectoration persist. At intervals, varying from a few days to a few weeks, catarrhal symptoms wax or wane, without any apparent cause. In these cases, influenzal bacilli continue to be found in the expectoration over long periods of time, in large numbers and in pure cultures. The general health, in such cases, is extremely variable. Sudden fever may occur, there may be bouts of perspiration, or there may be attacks resembling asthma.

I have seen several cases, in which the physical signs of consolidation have persisted for several months after an attack of influenzal pneumonia, and then have gradually disappeared.

Pfeiffer's bacillus was found by Netter in the expectoration during a period of 14 months in a case with induration of lung.

It should also be remembered that influenza may become chronic in patients suffering from pulmonary tuberculosis. Lesions, characterized by fibrosis with consolidation of large tracts of lung, may result from the chronic form of influenza. In such cases, bronchiectasis may follow later on.

An attack of influenza is given as the starting point in many cases of pulmonary tuberculosis. The patient, debilitated by a severe attack of influenza, has in his lungs a favourable soil for the tubercle bacillus, and offers a lessened resistance to its inroads. In the case of a patient with a quiescent tuberculous lesion, an attack of influenza appears to excite it to renewed activity.

TREATMENT.

The respiratory complications of influenza always add to the seriousness of the malady and to the anxiety and responsibility of the doctor. Every effort should be made to avert them. In the mildest attack of influenza the patient should at once go to bed, and remain there until some days after the fever has subsided. In the event of respiratory complications, the patient should, as a rule, be kept in bed until they have cleared away.

As we have no specific remedy, treatment has to be symptomatic. The cough is the first symptom of importance which demands our attention. The patient may be given barley water, linseed tea, toast and water, or warm milk to sip. Steam inhalations may be employed, such as the well-known Vapor Benzoini, or an inhalation of eucalyptus and menthol (Menthol xx gr., Tr. Eucalypt. Fol. ℥i. ℥i. to a pint of warm water). Small doses of heroin hydrochloride, $\frac{1}{36}$th to $\frac{1}{12}$th of a grain, may be given at intervals of one to two hours. When the cough is frequent, dry, and hacking, a sedative linctus may be tried

like the following :—

℞ Morphin. Hydrochlorid. - - - gr. ½.
Heroin. Hydrochlorid. - - - gr. ½.
Apomorphin. Hydrochlorid. - - - gr. ¾.
Acid. Hydrochlor. dil.- - - ♏ xx.
Syrup. Limonis - - - - ℥ ss.
Aquam Chlorof. - - - ad ℥ ij.

Ft. linct. Sig. ℥ i occasionally.

When there is bronchitis a mixture, such as the following is useful :—

℞ Liq. Ammon. Citrat. - - - - ℥ jss.
Pot. Citrat. - - - - - gr. xv.
Vin. Ipecac. - - - - - ♏ x.
Aq. - - - - ad ℥ i.

T. d. s.

An ammonia and nux vomica mixture with squill or senega may be given later if secretion becomes profuse. When the secretion is thick, viscid, and expectorated with difficulty, preparations containing opium are contra-indicated and should not be given.

Linseed or linseed and mustard poultices, applications of antiphlogistine, or turpentine stupes may be employed, when there is tightness of the chest, or soreness or pain on breathing.

The same remedies may be used in cases of broncho-pneumonia, but in this complication, as well as in lobar pneumonia, the main indication is to keep a watch on the heart.

The chief danger in influenzal broncho-pneumonia and pneumonia is cardiac failure. Every effort should be made to maintain the patient's strength. When there are signs of failing heart, stimulants are of special value. Of these the most important are alcohol, strychnine, caffein, camphor, digitalis, and strophanthus. Alcohol is indicated when there is prostration, and specially in cases in which there is delirium and sleeplessness. Champagne and brandy are the best forms in which to administer it. The amounts to be given must be regulated by the age and condition of the patient, and by the effects on the heart and pulse in individual cases. The mistura spiritus vini gallici is an excellent combination of nourishment and stimulant.

Strychnine is of great value in failing heart, and should be administered hypodermically in doses of from $\frac{1}{60}$th to $\frac{1}{20}$th of a grain, being repeated as required at intervals of three or four hours. Caffein is another cardiac stimulant in which I have faith, and it also is best administered hypodermically in three or four grain doses.

Intramuscular injections of camphor dissolved in olive oil have come much into favour in this country of recent years, but have long been advocated on the Continent as cardiac stimulants by Finkler and others. The ordinary dose is 2 to 3 grains of camphor dissolved in pure olive oil (1 in 10). It may be obtained ready for use in the form of ampoules. Camphor may also be given as a stimulant by the mouth, 5 drops of Rubini's solution on sugar repeated from time to time. Huchard and other French physicians recommend 5 minim doses of the 1 in 1,000 solution of crystallized digitaline (Codex Suppl.).

Digitalis is a remedy which has long been advocated in the treatment of pneumonia, and no doubt it is of value, as Wilson Fox maintained, in cases in which from an early period there is undue frequency combined with weakness of the pulse, or when the expectoration is profuse and bloody, and in cases of alcoholism when there is profuse sweating; but it should be given early in these cases, and combined with alcoholic stimulation. Wilson Fox believed, however, that it was contra-indicated and even dangerous in old people. I specially mention digitalis here, because I find that some valuable old remedies are apt to be lost sight of at the present time.

Strophanthus is another valuable cardiac stimulant. The best form to employ is hypodermic or intravenous injections of strophanthin, dose $\frac{1}{500}$ to $\frac{1}{100}$ grain. The latter is the better when rapid effect is desired.

An old-fashioned remedy which I have sometimes employed, I think, with advantage in cases of prostration in elderly subjects is musk. Five to ten grains may be given in a cachet, or it may be prescribed in the following form :—

℞ Moschi - - - - - gr. iij.
Sp. Menth. Pip. - - - - ℥ xv.
Sp. Ammon. Aromat. - - - - ℥ xlv.

Ex. aquam. p. r. n.

Or, as Sir Dyce Duckworth advises, combined with Hoffman's Anodyne, as follows :—

℞ Moschi - - - - - gr. v.
Sp. Aetheris Co. - - - - ʒ ss.
Mucilaginis - - - - - ʒ i.
Aq. Aurant. Flor. - - - - ʒ i.

Ft. haust. p. r. n.

Sleeplessness is sometimes a prominent symptom and, when combined with delirium and prostration, a cause of great anxiety. Opium and morphia are usually contra-indicated, on account of drying up the secretions and clogging the air passages, and great caution is needed if they are administered. Probably one of the best combinations to employ is chloral and potassium bromide, 15 grains of the former with 30 grains of the latter. Chloralamide has been regarded as safer than chloral, but it is not so effective in producing sleep, and lately it has not been obtainable. Other hypnotics, such as paraldehyde, sulphonal, trional, dial, and veronal, may be employed, but I have found them all uncertain in their action in the sleeplessness of pneumonia. Whatever hypnotic is employed, alcohol should also be administered.

Sponging, the application of cold compresses, or the wet pack, may be used in cases of sleeplessness or when the temperature is high.

Oxygen inhalations should be employed when there is dyspnœa and cyanosis; the rate of the respirations and the colour of the lips

and extremities will be the best indications or their employment. They should be given for such times and at such intervals as the condition of the patient demands, but should not be given continuously unless the condition of the patient is desperate. During the present epidemic there has been a great difficulty in obtaining a supply even for our hospital cases. One has had, therefore, to be sparing of its use.

The diet should consist mainly of milk, beaten-up eggs, beef tea, chicken broth, rusks, Benger's food, calf's foot jelly, etc. At the present time, beef tea and chicken broth are likely to be almost unobtainable, and the diet may be supplemented by Bovril, Glaxo, Brand's Essence, Valentine's Meat Juice, etc. Often there is an utter distaste for food. The mouth, the tongue, and the teeth should be kept as clean as possible. The action of the bowels should be carefully attended to.

The room in which the patient is nursed should, if possible, be large and airy, but he must be kept out of the way of draughts. If there is bronchitis, screens round the head of the bed and a bronchitis kettle are advisable. At this time of the year a fire should be kept up in the room day and night, but the room should not be allowed to get too hot. A temperature of about 62° F. is best for the patient. With a fire in the room the window can be left open. Purity of the air of the sick room is more important even than its temperature; but one must use common-sense in these matters and avoid extremes in both directions.

The best prophylactic against influenza and its respiratory complications is fresh air. Nothing is more striking than the manner in which influenza spreads in stuffy and ill-ventilated hotels. Sir Dyce Duckworth has called attention to the risks of picking up influenza in trains. We are far in advance of our continental neighbours, friends and others, in our love of fresh air, but there are still far too many who regard an open window as a source of danger, and insist on closed windows in trains, hotels, and all places of public resort. Fresh air is the best disinfectant, but it must also be remembered that where there is dust or dirt there is danger. The interiors of houses, shops, offices, workshops, theatres, meeting halls, churches, and public conveyances, must be kept clean as well as airy if they are to be safe and free from the germs of such a disease as influenza and its complications.

VACCINE TREATMENT.

It has often been pointed out that influenza differs from other infectious fevers in the fact that one attack does not lead to immunity from others. Persons who have had one attack indeed seem to be rather more than less susceptible when exposed to infection. If the influenza bacillus is the cause of the malady, there would be no theoretical reason for expecting a vaccine prepared from it to confer any immunity of more than short duration; but on the supposition that the true virus is still undiscovered, we may still be able by means of a vaccine to protect the body from the allies, which are several

times more dangerous than the virus itself. A vaccine of this kind has been suggested by a committee of expert bacteriologists for tentative use in the Army. Two prophylactic inoculations were recommended. The first was as follows: B. influenzæ, 30 millions; pneumococci, 100 millions, streptococci, 40 millions. After 10 days a second was given twice as large as the first. Exception was taken to this by more than one authority on the ground that the dosage of influenza bacillus was too small, and that to be effective a dose of 200 or 300 million B. influenzæ should be employed. It was suggested that, in the treatment of chronic cases of secondary infection, doses of one fifth the first dose might be employed. The use of a vaccine in severe secondary broncho-pneumonias was not recommended, but one-twentieth of the first dose was suggested as a suitable dose. Vaccine treatment must at the present time be considered as only on trial, and a matter of theory rather than of proved utility.

The treatment of pleurisy needs a word in conclusion. The pain of dry pleurisy may be relieved by local applications, such as belladonna liniment, fomentations, or dry heat. When there is effusion, the possibility of an empyema must be borne in mind, and, if found to be present, should be treated surgically.

INFLUENZA AND THE USE OF VACCINES.

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The Controlling Editor has kindly given me considerable space in order that I may bring up to date, and considerably add to, my article published in the previous Special Influenza Number of The Practitioner in 1907.

The bacteriology of the epidemic of influenza which occurred in the early nineties, coming as it did in a period of great activity in pathological research, was naturally the subject of careful study, and the discovery of the micro-organism which appeared to cause the disease was first announced by Pfeiffer in 1892, and soon corroborated by numerous observers in all parts of the world. The *Bacillus influenzæ*, or Pfeiffer's bacillus, is now well known, though the difficulties attending its cultivation have hitherto prevented the acquisition of such a complete knowledge of its characteristics as is obtainable in the case of most of the other important pathogenic bacteria. It is extremely small, rarely measuring more than $\cdot 5\ \mu$ in length, *i.e.*, it is roughly only one-sixteenth of the diameter of a red corpuscle long. It forms no spores, has no flagella, and is non-motile. It does not stain by Gram's method, and requires the use of rather powerful dyes for its demonstration. When properly stained it often shows a cap of colour at each end with a clear space intervening. This "polar staining" may lead to its being mistaken for a diplococcus if carelessly examined.

The main cultural characters of Pfeiffer's bacillus are these: It is a strict aerobe, requiring the presence of oxygen for its activity; it grows best at the body temperature, and not at all below 27° C. or above 42° C.; and it refuses to grow except on a medium containing hæmoglobin.* The most convenient medium for its cultivation is ordinary agar which has been streaked with a little blood; this may be taken from an ordinary skin-puncture, provided that careful precautions as to asepsis are preserved, and that the medium is inoculated for a day or two before being used, so as to ensure sterility. When grown in this way, it forms very minute transparent colonies, which look like drops of dew. Even in the most favourable circumstances it grows feebly and dies out in a few days if it is not frequently transplanted. Involution forms (threads, chains, etc.) are frequently formed in cultures.

A point of considerable theoretical interest has recently attracted

* This is not strictly true, for it may be grown with some difficulty on other media containing certain animal substances (Cantani and others).

some attention. This is the ease with which the influenza bacillus will grow without hæmoglobin, provided that certain other bacteria accompany it. This was first pointed out by Cantani, who succeeded in obtaining cultures when the organism was inoculated on ordinary media along with diphtheria bacilli, staphylococci, and other organisms, and found also that the latter would act in this way even after they had been killed by heat. Luerssen[1] found that the most suitable " nurse " for the influenza bacillus was the *B. prodigiosus*, and added the interesting fact that cultures of this bacillus, killed by being heated to 60° C. retained this action, whereas those heated to 100° C. were inert. Neisser[2] recommends the use of the xerosis bacillus, and succeeded in keeping the influenza bacillus alive for twenty generations when the two were grown symbiotically. In some cases the latter organism was found to be still living when the cultures were twenty days old. There are also some reasons for thinking that killed cultures of streptococci may increase the virulence of the bacillus for animals. If this should prove to be the case it opens up an interesting field for speculation as to the origin of the epidemics of influenza, suggesting that Pfeiffer's bacillus may live, possibly for long periods, as a harmless saprophyte in conjunction with other bacteria, and be suddenly raised in virulence as a result of its entrance into the body in conjunction with the streptococcus, or some other pathogenic bacteria. As we shall see, bacilli which, if not actually identical with Pfeiffer's bacillus, have the closest possible resemblance thereto, are widely distributed, and produce diseases which are constantly present; and it is easy enough to imagine that these might receive, in some such fashion, the slight alteration in their metabolism necessary to transform them into the pathogenic bacillus which occurred in the pandemic in the nineties.

The pathogenicity of the organism for the lower animals is but slight, and the effects following its injection are not similar to influenza in man. Injected into the peritoneal cavity of guinea-pigs in rather large doses it causes acute fatal peritonitis. Animals can be immunized against it, and the immunity is said to depend on the presence of a bacteriolysin, but the common presence of the organism within the leucocytes in the sputum in influenza would suggest that an opsonin plays an mportant part in this respect. The immunity left in man by an attack of the disease is of short duration.

Several experimenters have produced sera against this infection. In some cases, these were of some value in experimental infections, but I do not know whether they have been tried clinically.[3] [4]

The pathology of the disease is not fully known, the rarity of autopsies during the acute stage rendering its investigation difficult. In most cases the lesion is limited to the respiratory mucosa, sometimes the cavity of the nose, and occasionally only the tonsil[5] being inflamed, though usually the disease extends to the bronchioles, or alveoli of the lungs. More rarely other regions, such as the conjunctiva and (in one case at least) the urethral mucosa, are affected primarily.[6] The bacillus can usually be demonstrated with ease in

the secretion from these lesions, the simplest method being to spread a thin film, dry, fix, stain deeply with carbol-fuchsin, aiding its action by gentle heat, wash thoroughly in a stream of water, and finally to dry and examine with a high power. In such preparations Pfeiffer's bacillus may often be seen in pure culture; frequently, however, other organisms are present, and there is no doubt that secondary or concurrent infections, though not necessary, play an important part in the pathology of the disease. For example, the pneumonia of influenza may be due to Pfeiffer's bacillus alone, or to this organism in conjunction with the pneumococcus, *M. catarrhalis*, streptococci, or other bacteria. The inflammatory sequelæ of influenza (such as otitis media) are mostly due to these other organisms, especially pneumococci and streptococci.

In some cases the bacillus enters the blood-stream, converting the disease into one of septicæmic type; the frequency with which this occurs is somewhat doubtful. Thus Canon[7] found them in blood-films in all cases of the disease, whereas Klein[8] found them only in six cases out of forty-three, and other observers have not succeeded in finding them at all. Further, Klein failed to cultivate them from the blood in the six cases in which he found them in films, thus indicating that the disease is not of septicæmic type, and that the bacilli which gain access to the blood-stream are rapidly killed.

Horder[9] found living influenza bacilli in the blood in two cases of ulcerative endocarditis, thus proving that living bacilli may circulate in the blood, and become engrafted on distant organs. A case of peritonitis due to this bacillus has also been recorded.

The general and remote symptoms of influenza must doubtless be attributed to the toxin of the bacillus, whether produced locally, in the inflamed mucosa, or liberated by the solution of the bacilli in the blood-stream. It seems highly probable that the latter is an important source of toxin, and one which is not sufficiently recognized. It affords an explanation of the fact that the nervous and other symptoms of influenza are by no means proportionate to the severity of the primary lesion. Thus in the cases in which there are severe general and remote symptoms with very few signs of local respiratory or other disorder it is likely that many bacilli gained access to the blood, were destroyed, and liberated their toxins into the circulation.

The pathology of the gastro-intestinal and other atypical forms of influenza is unknown; the organism may, for all we know, attack the mucous membrane of the digestive tract, or the symptoms may be due to remote toxic action.

But little is known of the nature of the toxin of Pfeiffer's bacillus. Filtered cultures show no toxicity, and the endo-toxin has been investigated by Slatineano,[10] using Besredka's[11] method of dissolving the bacilli in fresh serum, and removing the bacilli by centrifugalization. When injected into the brain of a guinea-pig it kills in small doses, but this has little action in the peritoneum.

So far no serious difficulties have arisen. Observations, however, since the earlier years of the epidemic, have shown that the facts are by no means so simple, two disturbing facts being brought to light.

These are, first, the existence of bacilli which are indistinguishable from those of Pfeiffer, but which do not cause influenza, and, secondly, the fact that a disease clinically identical with influenza may be caused by organisms other than this bacillus.

One of the first of the influenza-like bacilli to attract attention was that which occurs in abundance in the sputum in whooping-cough, and first observed by Sprengler and by Eppendorf in 1897. This organism has the closest possible resemblance to Pfeiffer's bacillus, the only difference being (according to some only of the observers who have investigated it) that it is slightly larger, a feature that depends on so many factors (especially on the staining method employed), that it is unsafe to attach much importance to it. The two bacteria are identical in their cultural characters, and both show the same symbiotic relations to other organisms. The effects of the inoculation of the whooping-cough bacillus on to the nasal mucosa of man (as described by Davis[12]) is at least closely similar to that of Pfeiffer's bacillus similarly applied, as described by Cantani.[13] And Wollstein[14] has recently shown that the serum of animals immunized against Pfeiffer's bacillus, agglutinates the *B. pertussis* and *vice versâ*, though the serum is more potent against the organism used for the injection. Taking all the facts into consideration we must come to the conclusion that there is no sufficient evidence to show that the organisms are not identical, and that it is a mere begging of the question to distinguish between a " true " and a " false " bacillus of influenza in the case of two bacteria which are indistinguishable, but which come from different sources.

Another important and widely distributed organism closely allied to Pfeiffer's bacillus, and possibly identical therewith (though the evidence is not so strong as in the preceding case), is the bacillus of epidemic conjunctivitis associated with the names of Koch and Weeks, and recognized before the discovery of the influenza bacillus. The bacillus described by Müller in trachoma is also very similar, and at least three other organisms which are identical in morphological and cultural characters have been described. Most writers have fenced with the question, and refused to give a decided opinion as to the identity or non-identity of these bacteria, but Iundell[15] has taken the logical step of asserting that they are all accidental modifications of the same species, and regards conjunctivitis due to the Koch-Weeks bacillus as a form of ocular influenza. This view would at least explain the presence of bacilli which are potentially those of influenza in the absence of an epidemic, though it would naturally leave us to seek the causes, whether symbiotic, atmospheric, telluric, etc., which led to its assuming such remarkable pathogenic properties.

The occasional non-occurrence of Pfeiffer's bacillus in disease clinically identical with influenza has been noticed, in all probability, by most bacteriologists during the last few years, and has been attributed to defective technique, unsuitability of the material examined, or to an incorrect diagnosis of the disease from which the patient is suffering, and such cases have not been recorded. Lately, however, the discrepancy has become very obvious, and observations

made in England, France, Germany, and America have shown that in attacks which are clinically typical influenza the bacillus may not be forthcoming. This was first pointed out in this country by Bulloch,[16] and since then Dunn and Gordon[17] have published an account of an " epidemic simulating influenza," the bacteriological aspect of which was studied with much care; the main feature was the absence of Pfeiffer's bacillus, and the frequent presence of *Micrococcus catarrhalis*, to be described subsequently. An even more interesting epidemic was investigated by Besançon and de Jong,[18] who found Pfeiffer's bacillus present in a few instances only, the pneumococcus almost constantly, and occasionally other organisms, such as the pneumo-bacillus, streptococci, pseudo-diphtheria bacilli, etc. In the majority of cases they also found *M. catarrhalis*, or an organism resembling *M. tetragenes*.

Other epidemics with similar bacterial associations have been described, but it is unnecessary to labour the point, as it must be perfectly familiar to all who practise clinical bacteriology at the present day. Pfeiffer's bacillus is still met with but it is comparatively rare, and the most usual organism to find in the sputum or nasal discharge of cases of clinical influenza nowadays is *M. catarrhalis ;* after that pneumococci, streptococci, pseudo-diphtheria bacilli, etc.

The *M. catarrhalis* was first described by Seifert in 1896, and was found soon after this by Pfeiffer in conjunction with his bacillus in the lungs in broncho-pneumonia in children. Since then it has gradually become well recognized as a cause of numerous forms of inflammation of the respiratory tract, varying from mild catarrh to severe broncho-pneumonia. It is a diplococcus, often of rather large size, which does not stain by Gram's method, and thus presents some resemblance to the gonococcus and to the meningococcus; when examined in films of pus the resemblance is heightened by the fact that the organisms are frequently intracellular. There is no doubt that confusion has arisen in the past, but with our present knowledge it is not difficult to discriminate between them. *M. catarrhalis* grows at the room temperature, thus distinguishing it from the other two. It forms greyish colonies, which spread somewhat, and does not liquefy gelatin. When cultures are examined the cocci are found to be variable in size and arrangement, diplococci being mixed with short chains, isolated forms, etc.[19]

Thus we see that what is apparently Pfeiffer's bacillus does not always cause influenza, and the influenza (regarded as a clinical entity) is not always caused by Pfeiffer's bacillus. What explanation are we to give of these facts? Two solutions present themselves—(1) that influenza is to be regarded as a specific disease due solely to Pfeiffer's bacillus, and that other disease clinically identical, but due to other organisms, must be called " pseudo-influenza " or some other question-begging name; or, (2) that it is not a pathological entity, but a group of diverse diseases due to various bacteria. There are serious difficulties connected with either explanation.

With regard to the former explanation, it must be pointed out

that, although causation is the soundest basis for nomenclature, we are not justified in separating diseases which are clinically identical into different groups unless we can show some good reason for doing so. In the present case the physician would hardly thank us if we told him that a patient in whom he had diagnosed influenza was not suffering from that disease because Pfeiffer's bacillus was absent, but was really ill of a disease identical in symptoms, course, event, sequelæ, and treatment. If we could offer him a potent serum in the " true " cases, the matter would be different, but at present no good would result from attempting to alter the nomenclature which has been accepted for centuries.

Further, if the diseases occurred in well-defined epidemics, which did not blend with one another, there might be some excuse for this view. But this is not the case. The organism, present in the earlier years of this epidemic, was Pfeiffer's, and it was usually pure or nearly pure. At a later period, this bacillus and *M. catarrhalis* were frequently found in association in the same patient, and in some of the most recent epidemics *M. catarrhalis* often occurs unmixed with other bacteria, and in the same epidemic some patients may show Pfeiffer's bacilli, some *M. catarrhalis*, others yet other bacteria. It is absurd to term some " true " and some " false " influenza.

We are led to the second possibility, that influenza is not a specific disease, but one which may be due to a variety of bacteria. This does not solve the difficulties, but raises fresh ones. For instance, how can we explain the fact that the toxins of these various organisms produce symptoms which are clinically indistinguishable? And these symptoms are not necessarily those which are caused by the organism in question when not associated with an epidemic of influenza; thus *M. catarrhalis*, in ordinary circumstances, causes mild broncho-pneumonia, which is much less severe than that of influenza.

But a more difficult question arises regarding its epidemicity. We may understand that *one* organism may suddenly become virulent, though we cannot explain how; but it is difficult to imagine how the numerous bacteria which we may find in influenza at the present day can have undergone this rapid change. It is a new feature in epidemic diseases.

The only possible solution that is entirely satisfactory is absolutely hypothetical and unsupported by any evidence. It is that influenza is a specific disease, due to a definite single cause, but that this cause is undiscovered, and perhaps unsuspected; it may be an " invisible " microbe, a protozoan, or some depressing " influence " acting directly on the human constitution, and of a nature as little known as were bacteria when influenza received its name.* We must assume that

* Influenza was formerly attributed to an excess of ozone in the air (Schönbein), to an excess of negative electricity, and to terrestrial magnetism. The first condition, if it occurred, might well injure the respiratory mucosa and allow access to various organisms. Sir Thomas Watson, writing in 1857, alludes to these theories as being more probable than the germ theory, then attracting attention. The chapter is worth reading.

this factor has some peculiar action on the body, causing definite symptoms, and has in addition the power of temporarily lowering its resistance to certain organisms. If Pfeiffer's bacillus is identical with the other members of the group of hæmophilic bacteria it is a widespread organism, and there is no difficulty in seeing how it might take advantage of this breach in the defences of the body; and the other organisms are quite commonly met with in the mouth, air, etc.

This view of the pathology of influenza is similar to that generally held with regard to scarlet fever. In the latter disease the cause is unknown, and the local lesions and complications are due to common bacteria acting on tissues of lowered resistance.

The views thus expressed twelve years ago still hold good at the present day; we do not know the cause of influenza, and the secondary infections are now realized to be of chief importance in the production of the mortality associated with the disease. It has taken a long time to get this view accepted. Thus, Muir and Ritchie, writing in 1910, referred to " the well-established fact that it (Pfeiffer's bacillus) is always present in the secretions of the respiratory tract in true cases of influenza," and Hewlett, in 1914, expressed the opinion that it " is met with in all uncomplicated cases of influenza in the nasal and bronchial secretions." At the present time, and especially in view of the bacteriological findings in the recent pandemic of the disease, probably hardly anyone really holds this view. Pfeiffer, it is true, is " still investigating " the question, and Uhlenhuth blames the young bacteriologists of the present day for not knowing how to recognize the bacillus in question, but we may state, without fear of contradiction, that there is now such a mass of negative findings, that the thesis that Pfeiffer's organism is the actual cause of all cases of influenza cannot be seriously maintained.

It is not that the organism in question never occurs. As a matter of fact it is probably the commonest microbe to be found in this present epidemic, both in the sputum and nasal discharge of mild cases and in the lungs in fatal cases of pneumonia; it is also found in the blood, though comparatively rarely. Two things, however, are quite clear, and either of them is sufficient to discount very strongly the ætiological part of the organism in influenza. The first is this: it occurs as an important infecting agent in diseases which are not clinical influenza. Thus, in an epidemic of " purulent bronchitis " occurring in a camp of New Zealand soldiers, Eyre and Lowe found it in 12 out of 14 sputa examined; the outbreak followed (in many cases) measles and rubella, and was evidently not considered by the authors to be influenza, since they tell us that in June an epidemic of this disease occurred, in which also they found Pfeiffer's bacillus as the prime ætiological agent. Similar experiences must have fallen to the lot of all bacteriologists, and instances of infection with this organism have occurred sporadically during the past ten or twelve years in cases, which present no clinical resemblance whatever to influenza as it occurs in epidemic form, and at places and in times

when the disease is unknown.

The other point is, perhaps, more important; it is that epidemics, which are recognized by competent observers as clinical influenza, have occurred and are occurring in which the bacillus is not found, or is only found in a small proportion of cases. In England not many cases have been reported in full, but several writers have given accounts of their findings, and in all, or nearly all, the influenza bacillus has been found frequently, but not always, which has been my own experience. But on the Continent a number of outbreaks have been described in which the organism has occurred but rarely, if at all. Thus Scheller described influenza in Leipsic as long ago as August, 1917, due to an organism which he described as *diplococcus mucosus*, and other epidemics were recorded during the same year. In Germany, the epidemic of influenza attained large proportions early in the year (1918), and much interest was taken in its bacteriology. The great majority of observers failed to find Pfeiffer's bacillus at all; this was the case with Kroner and Kisskalt in Kiel, Friedemann in Berlin, Kelle in Frankfurt, and Hirschbruch at Metz.* At Mainz, Gruber and Schadel found it 14 times in 250 cases, whilst at Marburg, Bergmann isolated it 15 times out of 20. Other cases from Continental writers might be quoted.

One of the most interesting epidemics in this connection was that which occurred in the United States in the winter of 1915 and 1916. According to Mathers, it "resembled in every detail epidemic influenza," and was different from common acute rhinitis. It was attended by a considerable mortality, so that the general mortality of the country was greatly increased. The important point about this epidemic is that the predominating organism was a *streptococcus*, an organism which had been often recorded as occurring in influenza before this, but never, so far as I am aware, so commonly, and this is of especial importance in that, in the severe cases which we see in England and in Europe generally, this organism is the most important, because the most fatal of the associated organisms. It does not occur in all cases, even in fatal ones: we still see pneumonia and bronchopneumonia proving fatal when only Pfeiffer's bacillus, the pneumococcus, and *M. catarrhalis* are present. In most cases, however, which tend to a fatal termination, and more especially in the fulminating or "septic" cases, streptococci form the predominating infection, and may be pure, or apparently so, both in the lungs and in the blood. As regards the nature of the streptococcus, not very much information is to hand at present; indeed, in the present unsatisfactory state of our knowledge of this organism, we can hardly expect it. In the severe cases the organism has usually hæmolytic powers, suggestive of high virulence; this was found by Mathers in America in 75·4 of the total number of cases, a proportion which is probably not attained in the outbreak as we see it here. Mathers also

* Vide *Medical Supplement to the Daily Review of the Foreign Press*, October 1, 1918.

found the less virulent " viridans " type, which forms a green colour on hæmoglobin media, in 49·7 per cent. of cases.

In addition to these streptococci which, from their high pathogenic powers, constitute so important a feature in this present outbreak, it seems to me certain that pneumococci are more prevalent than in former epidemics. Mathers found them in 49·1 per cent. of his cases, but not usually pure, and he adds the interesting and important fact that the biological forms, which were most common in ordinary lobar pneumonia, were not those which predominated in the epidemic; thus in the latter the pneumococci of Group IV. predominated, in the former those of Group I.

Another interesting argument should be mentioned. The incidence of the disease in this epidemic is especially on comparatively young patients, and, in contrast to the epidemic of the early nineties, old people are not especially affected, nor is the mortality amongst them very high. This has been thought to indicate that we are dealing with a new disease, but by far the more likely explanation is that those who were affected in the former epidemic are immune, and it is in the new generation who were not exposed to the disease that we now see it in such abundance. An interesting point, as tending to show the identity of the two diseases is that organs of patients who had died in 1890–1892 from the epidemic of that period, preserved in museums, show an essential similarity with those of patients dying at the present day. The clinical differences which we see in some cases are not more than we might expect from differences in the secondary infections.

Now if this is so, the case for regarding Pfeiffer's bacillus, or any of the ordinary organisms of catarrh as the cause of true influenza disappears altogether. The influenza bacillus does not cause a short, sharp infection with complete and permanent subsequent immunity. There is experimental evidence to show that it is extremely difficult to immunize animals to its action, and in man it often causes chronic infections, which may last for months or years, or which may often relapse; the same is true of the pneumococcus, streptococcus, and the other organisms often found in this disease. Looking at the matter from the point of view of the long-lasting immunity, everything seems to point to the fact that we have to deal with one of the diseases of unknown origin similar to scarlet fever, small-pox, measles, etc., in which the immunity is usually life-long, rather than with a bacterial infection, in which it is transitory; and influenza, apart from its secondary complications, is a self-limited disease, which is rare in the case of bacterial infections and common in the exanthemata of unknown origin.

This, then, is the position at the present time. Influenza bacilli are probably the most usual organism found in cases which are undoubted epidemic influenza; but they occur also in cases which no one would think of calling influenza if it were not for the presence of the bacillus. Apart from this, the pneumococcus is probably the most frequent, and the streptococcus the most fatal; but other

organisms, such as *M. catarrhalis* and other diplococci, not yet fully described or identified, are not uncommon. If we still hold that Pfeifler's bacillus is the cause of influenza, we must admit that it is impossible to diagnose the disease clinically, even in an epidemic form, and also that influenza may occur, apart from an epidemic, in forms having not the slightest resemblance to the disease we know so well. This appears to make confusion more confounded; the view expressed in the earlier article appears to meet the indications perfectly at the present day, and, just as the unknown virus of scarlet fever renders virulent the streptococcus of the mouth, so we must assume that the hypothetical virus of influenza can render virulent streptococci, pneumococci, or other pathogenic organism which may be at hand. In their absence, or in the case of a patient who is immune to their action, the disease caused by this hypothetical infection will be mild; if it opens the door to their action, and if the patient is unable to resist them, an infection almost as rapidly fatal as any we know will occur. It is a possibility that the streptococcus which has formed so dramatic a part of the present infection is of American origin, and it is an interesting fact that the Germans attribute their epidemic to the American troops. According to them it first appeared in a battalion which had been fighting against them, and spread subsequently to other troops, and to the civil population. This, however, is quite uncertain in view of the earlier epidemics of influenza-like disease which had occurred; but it is quite possible that the particular strain of streptococcus is a newcomer as a secondary infection.

It must be realized that if this theory of influenza is true, the secondary infections which constitute the most important part of the disease may be a patient's own, or may be derived from another person. If the disease produces the great fall in immunity which we have suggested, then any virulent microbe which he may harbour in his mouth, etc., and which has hitherto been a mere harmless saprophyte, may attack him. On the other hand, when he is infected by drops of sputum coughed out by an infected person, he may easily be infected simultaneously by the hypothetical influenza germ, and by another organism, perhaps of exalted virulence from "passage" through the body, against which his resisting powers are slight or almost absent. In this way epidemics in different parts may be very different in clinical features, the predominating secondary infections being different; even in the same epidemic and in the same neighbourhood, the chief secondary infections may be (and we know they are) different in different cases.

The importance of all this arises mainly from the highly practical question of the use of vaccines as preventive and as curative measures. As regards the first question, it is hardly necessary to point out that if the influenza bacillus is not the cause of the disease, we can scarcely hope to get good results from the use of a vaccine made from this organism as a prophylactic measure; it is, of course, remotely possible that the cause of the disease is an organism which will grow symbiotically on our media along with the bacillus, so that we may

actually be administering it when we give our vaccine, but this is hardly to be hoped for. On the other hand, by a suitable admixture of the secondarily infecting organisms we may hope to diminish greatly the severity of the disease, by reducing it to influenza alone, minus the other organisms, which are usually the fatal infections.

The only way in which this question can be answered (it is one that every bacteriologist is being asked, almost every hour of the day), is by the results of practical experience on a large scale, and this is not yet forthcoming. The only figures which are known to me as having any bearing on the question are those quoted by Eyre and Lowe. They were called in to deal with an outbreak of purulent bronchitis occurring in a camp of New Zealand troops, which was attended by a high mortality, and in which the predominating organisms were *B. influenzæ*, *streptococcus longus*, *M. catarrhalis*, pneumococcus, and sometimes other organisms. This was combated by prophylactic inoculations of a highly complex vaccine, containing pneumococci, streptococci, *B. influenzæ*, *staphylococcus aureus*, *M. catarrhalis*, *B. pneumoniæ*, and *B. septus*, with apparently good results. This was first given in March, and in June the epidemic of true influenza occurred, and the figures seem to show that the numbers of uninoculated men who were affected were very much greater than those of the inoculated. Unfortunately, however, the figures are not given in such a way as to lead us to get a very clear idea of the comparability of the inoculated men and the controls, nor is it obvious whether the figures quoted (2 per 1,000 amongst inoculated men and 28·4 in the uninoculated) refer to all cases, and, if so, over how long a period, or refer to hospital admissions only. It is much to be regretted that further statistical details were not given, for the possession of clear proof that the vaccine did actually diminish the incidence of the disease would greatly strengthen our hands in recommending its use.

The question has recently been put on a different footing by the issue of a report by an influential committee assembled under the auspices of the War Office. This committee agreed that there was considerable doubt as to the primary significance of the *B. influenzæ*, but thought also that a vaccine containing this organism as well as those causing the most important secondary infections—streptococci and pneumococci—might control the incidence as well as the severity of the disease. It is, as Sir Thomas Horder pointed out, a little difficult to understand how the vaccine in question is likely to influence the incidence of a disease when it does not contain the causative organism; however, we have now an official pronouncement which may serve in lieu of definite statistical evidence when we wish to urge the use of the vaccine as a preventive measure and have no observed facts to urge as proofs of its value and innocuousness.

The vaccine which is recommended contains, for the first dose for adults, 30 million influenza bacilli, 100 millions of pneumococci, and 40 millions of streptococci. No recommendations were made as to the race or type of organism to be used, except that the various

cultures should have been recently isolated from cases occurring in the present epidemic. Why *M. catarrhalis* was not included is not clear; it certainly occurs in the lungs in vast numbers in some fatal cases of broncho-pneumonia, and I know of no reason to think that it is not a secondary infection of considerable importance.

The second dose is to be twice as large as the first, and is to be given after an interval of ten days.

These recommendations have not escaped a certain amount of criticism. John Matthews, whose method for the isolation and cultivation of the influenza bacillus and whose experience with vaccines made therefrom give him a right to express an opinion, holds that the dose of the bacillus recommended is much too small, and that to a degree seriously impairing the value of the vaccine; he recommends a dose of 150 millions for the first injection and 300 millions for the second. In this he is supported by Sir Thomas Horder, who suggests that the cultures grown on blood agar according to the old technique are much more irritating than those cultivated on Matthews's trypsinized blood. This seems quite possible, and in any future recommendation of a vaccine made of this bacillus it would be desirable that the nature of the medium on which it is to be grown should be indicated.

With regard to the dose of pneumococcus also there is a serious difference of opinion. W. H. Wynn, whose experience of the treatment of pneumonia by vaccines is probably unsurpassed in this country, holds that they are much too small. His criticisms apply mainly to the use of vaccines in treatment, but with regard to prophylaxis he points out that Sir Almroth Wright gave 1,000 millions of pneumococci, whilst Lister used much larger doses. Here, again, the question of the culture medium may come in, for I have certainly seen very decided reactions after 50 millions of pneumococci. Very much more information is required on this, as on most subjects in medicine.

As regards treatment, the committee seem somewhat unduly cautious and pessimistic. In severe cases of secondary broncho-pneumonia, *i.e.*, when it is most required, they express the opinion that the use of the vaccine cannot be recommended, in view of the dangerous rapidity and severity of these cases, but when it is tried they suggest that the initial dose should not exceed one twentieth of the first dose described above, that is, 1½ millions *B. influenzæ*, 5 millions pneumococci, and 2 millions streptococci. Many bacteriologists hold that it is especially in these acute cases of broncho-pneumonia that the use of vaccines is most strongly indicated, always assuming that the treatment can be begun early. Thus Wynn urges the immediate treatment of influenza by giving adequate doses within a few hours of the onset; the dose that he suggests is 50 million pneumococci, 50 million streptococci, and 30 million *B. influenzæ*—how cultivated is not stated. This probably represents the best we can do for the patients, and there seems to be sufficient evidence available to justify us in the belief that patients who have

had a dose of vaccine at the very beginning of the attack usually escape without severe symptoms. Further than this it is not necessary to go at the present time. Where expert bacteriological help is at hand, but little time is lost by examining film-preparations from the sputum, and giving suitable vaccines accordingly, but this should never be done if it leads to any considerable delay.

The committee suggests that the vaccine is most likely to be of use in the treatment of subacute and chronic cases, and recommends beginning with a dose of one fifth of the initial dose, *i.e.*, of 6 million influenza bacilli, 20 million pneumococci, and 8 million streptococci. Here, again, the dose, certainly of the first two ingredients, would probably be regarded as much too small by most bacteriologists, and certainly 50 million pneumococci is a reasonable beginning dose in most cases.

Here I must conclude. The position of affairs at the present day is most unsatisfactory, and it is urgently to be hoped that properly organized research on a wide basis may throw some light on the matter before the next epidemic. At present, we know really but little more than our ancestors did, when they attributed the disease to an " influence " from the heavenly bodies and named it accordingly.

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THE TREATMENT OF INFLUENZAL BRONCHO-PNEUMONIA, WITH SPECIAL REFERENCE TO VACCINE THERAPY.

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Reduced to its simplest terms, the treatment of an acute infectious disease of bacterial origin resolves itself into providing or enabling the patient to provide sufficient antibodies to overcome the bacteria and their toxins. We know of no drug which, given in therapeutic doses, will destroy bacteria within the living body. The only examples of specific drug therapy concern the protozoal infections, such as syphilis, malaria and sleeping sickness. No doubt in the future synthetic chemistry will furnish us with drugs having a similar action upon bacteria. The discovery of the specific action of ethyl-hydrocuprein upon pneumococci foreshadows this.

With such a disease as influenza, the usual course is to leave the patient to develop specific resistance to his infection as best he may, and, without direct intervention in the struggle, to adopt merely expectant treatment, which should be directly aimed at placing the patient in the best position for the fight, giving those drugs, if any, which will support him until he succeeds or fails in the venture, and especially withholding those measures which would hinder his immunizing reactions. These may be truisms, but it must be confessed that much treatment of pneumonia is without conscious aim, is merely traditional and without rational basis.

If expectant treatment was always successful, specific treatment would not be required. But, faced with the tragic loss of healthy young men and women with whom expectant treatment should have had its best chance, we must ask ourselves whether we could not wisely intervene in the struggle and give direct aid. The only specific treatment available is with vaccines, and I hope to show that encouraging success has been obtained by their use.

But with vaccine treatment as with expectant treatment, certain preliminary measures are fundamental and must be carried out promptly and efficiently. Too often the enemy takes the outposts and front-line trenches before action begins. One advantage of vaccine therapy is that it is offensive, not merely defensive, and may compel immediate retreat.

Before all things I would place absolute rest and free ventilation. With the scarcity of nurses the majority of patients are nursed by unskilled friends, and the doctor must give exact instructions, preferably in writing. It is not sufficient to see the patient in bed; the bedpan must be insisted upon, unnecessary movements in bed and unnecessary talking must be forbidden. Even trained nurses do not

always appreciate the need for strictness, and there is often too much effort caused by routine washings and rubbings, reaching for the sputum-mug, and sitting up to cough which might be saved the patient. One garment should be worn, and much exertion is saved by slitting it down the middle. One too often sees a struggle to get nightshirt and vest over the arms and head for the purposes of examination.

Windows should be open widely night and day, and the bed should be in the centre of the room, unscreened. The temperature of the room should not exceed 60°. An open fire aids ventilation and is useful for the disposal of rubbish. Old-fashioned gas fires are definitely bad, but the modern radiant gas fire is unobjectionable. The patient's face should be protected from the direct rays from the fire. When first seen, the patient is usually too warmly clad and with too heavy bedclothes. Patients and their friends are still imbued with the notion that pneumonia is caused by cold and must be dispelled by heat. Recently I saw a young woman with a temperature of 105°, in a stuffy room of 70°, three feet from a large coal fire. She was clothed in woollen combinations, a thick vest and flannel nightdress, to which the nurse had added a Gamgee jacket. Four blankets and an eiderdown quilt formed the bed coverings. Avoid extremes of heat and cold. The patient should feel that the surroundings are pleasantly cool. Sheets next the patient are more comfortable than blankets and more likely to aid sleep. Gamgee jackets are in fashion, and the nurse will often put one on unless definitely told not to. It is difficult to understand the reason for wrapping a feverish patient, whom we wish to lose heat, in cotton wool. If the patient is propped up, a light flannel jacket over the shoulders is advisable. These details must be attended to by the doctor and not left to the discretion of friends.

A useful routine *diet* is of milk (citrated if there is vomiting) and albumen-water, 5 oz. of each alternately every two hours. Patients are usually thirsty, and will take abundance of plain water or lemon water. But, unless insisted upon, the need of water is usually overlooked. Many persons still think that it is dangerous for a feverish patient to drink plain water, and this notion must be overcome.

Unless definitely forbidden by the doctor, the patient is sure to be given beef-tea, especially one expensive brand which contains three parts of coagulable protein in 1,000. Beef-tea and chicken-broth should have no place in the treatment of pneumonia. But, for the sake of the flavour, a teaspoonful of beef-tea may be added to the albumen-water.

Regular attention must be paid to the hygiene of the mouth. Tooth-plates must be removed. The mouth should be wiped out every two or three hours with a rag moistened with a dilute watery antiseptic. Mouth washes containing glycerine are objectionable. A little oil placed in the nostrils or a liquid paraffin spray helps the patient's comfort.

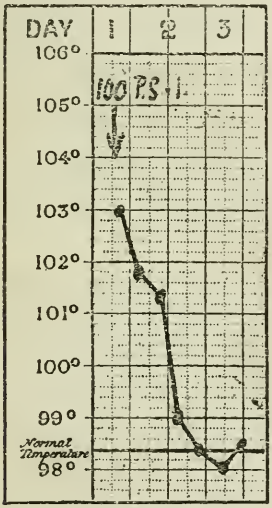
The general environment now being such that the patient can

make the best use of immunizing stimuli, the question of specific treatment can be considered.

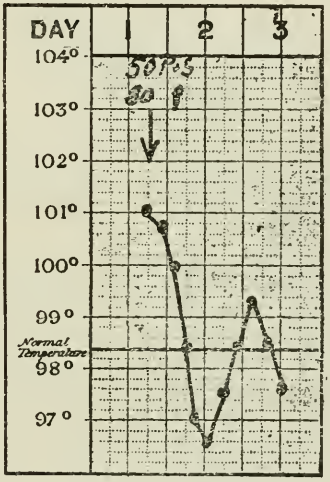
Broncho-pneumonia may develop in a patient as an after-complication, or it may be present from the beginning. As cases beginning mildly may later develop serious pulmonary signs, vaccine treatment, if it can be shown to cut short a simple acute influenza in its early stage, would prevent much broncho-pneumonia, and have a wide field of usefulness. My experience leads me to believe that this can be done, and that prompt treatment with vaccine within a few hours of the onset will definitely abort an attack of influenza. The following are illustrative cases.

SIMPLE ACUTE INFLUENZA.

CASE 1.—A man, aged 41. About 6 p.m. one evening he began to feel unwell, with shivering, headache, intense aching pains, nausea and slight vomiting. At 10 p.m. his temperature was just over 103°. He was given an injection of 100 millions pneumococci, 100 millions streptococci, 100 millions influenza bacilli. After an uncomfortable night his temperature next morning was 101°·8, and it rapidly fell to normal, which it reached 24 hours after the first symptoms began and 20 hours after the injection.



CASE 1.—*Acute Influenza.*
Male. Aged 41.

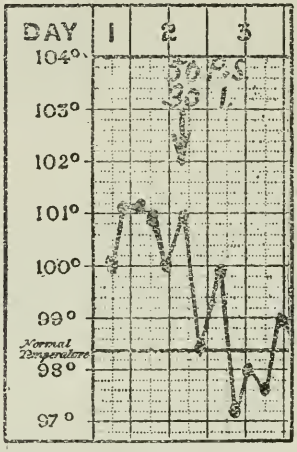


CASE 2.—*Acute Influenza.*
Female. Aged 26.

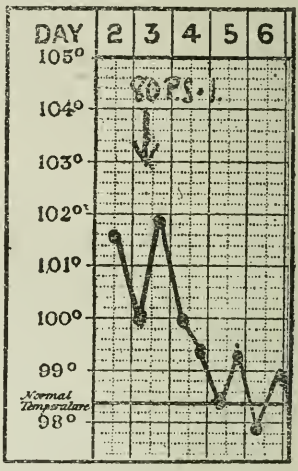
CASE 2.—A young woman. Onset rapid, with the usual symptoms. Temperature 101°, pulse 96, respirations 30. An injection of 50 millions pneumococci, 50 millions streptococci, and 30 millions influenza bacilli was followed by a fall of temperature, which reached normal in less than 12 hours after the vaccine. There was a slight rise to 99° the next evening, and thereafter uninterrupted recovery.

CASE 3.—A young woman, aged 29, was not injected until 24 hours after

the onset. There was then much headache and distress, and sticky crepitations were heard at the bases of both lungs. An injection of 50 millions pneumococci and streptococci, 30 millions influenza bacilli, was given. The temperature, which had previously ranged between 100° and 101°, fell to normal and, after a swing next day to 100°, remained down.



CASE 3.—*Acute Influenza.*
Female. Aged 29.



CASE 4.—*Acute Influenza.*
Male. Aged 20.

CASE 4.—Man, aged 20. Well marked symptoms, much headache and nausea, crepitations at both bases, irritating cough. Injected on third day with 80 millions pneumococci, streptococci and influenza bacilli. This was followed by a fall of temperature from 102 to normal.

These cases are in no way exceptional, and are given merely as representative of what may be expected of vaccine therapy in simple acute influenza. Other charts showing the effect of vaccine at a later stage could have been given. In all cases the effect was the same. After a period of sustained fever, the injection of vaccine was followed by a rapid fall to normal. When injected later than the second day, it is difficult to say that the temperature would not have fallen if vaccine had not been given, but in all cases there was an almost immediate change in the type of temperature. Either it falls by crisis and remains normal, or after falling to normal the temperature previously sustained at a high level swings once or twice before finally settling. This change from a sustained temperature to a remittent one is a result of vaccine action, which I pointed out in connection with the treatment of septicæmias[1] as a sign of recovery beginning. But in cases injected within the first 24 hours there is little doubt that the disease was definitely cut short. I have seen no similar charts in untreated cases, and no untreated case in which the pyrexia lasted only 24 hours or less. Rarely the pyrexia is over in two days; more often it lasts four to seven days. In some cases I have given a second

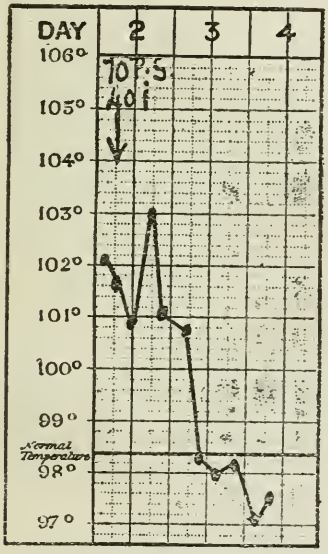
dose of vaccine after the temperature has reached normal. In none of these was there any temperature reaction, and a more rapid restoration to health apparently occurred. If further experience shows that vaccines can be relied upon to cut short the simple influenzal attack, we shall have the means in our power of preventing many cases of broncho-pneumonia.

In the cases in which broncho-pneumonia is present from the first, vaccine seems often to have the same good effect when it is injected early. I have not used it in the very severe fulminating cases, which are overwhelmed with toxæmia from the beginning. But many of my cases have been very severe and a few desperate.

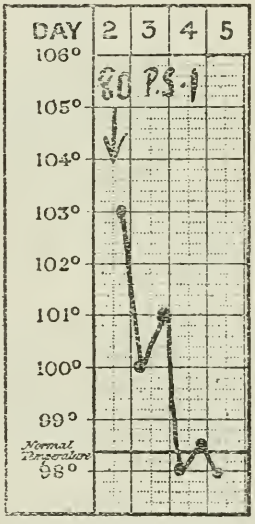
SEVERE BRONCHO-PNEUMONIA.

Case 5.—Man, aged 38. Admitted to hospital on second day of illness. Then had extensive signs of acute congestion at both bases, with cyanosis and red, frothy sputum. He was injected with 70 millions streptococci and pneumococci, and 40 millions influenza bacilli. Temperature, after a slight rise, fell, and reached normal next day. The pulse at same time fell from 96 to 68, and respirations from 28 to 20.

Case 6.—Man, aged 24, with well marked signs at bases, slight cyanosis, distressing cough, pulse 120, respirations 32. He was injected within 48



Case 5.—*Influenzal Broncho-pneumonia. Male. Aged* 38.



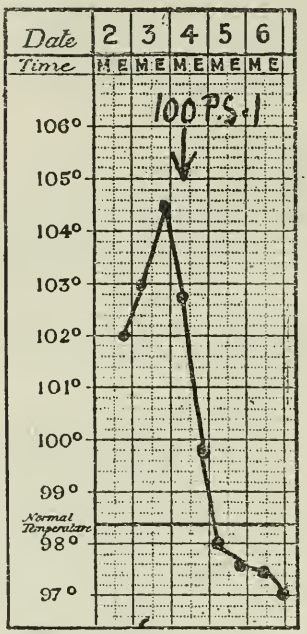
Case 6.—*Influenzal Broncho-pneumonia. Male. Aged* 24.

hours of onset with 80 millions streptococci, pneumococci and influenza bacilli. Temperature fell to 100° and, after a rise of one degree, fell to normal about 36 hours after the injection.

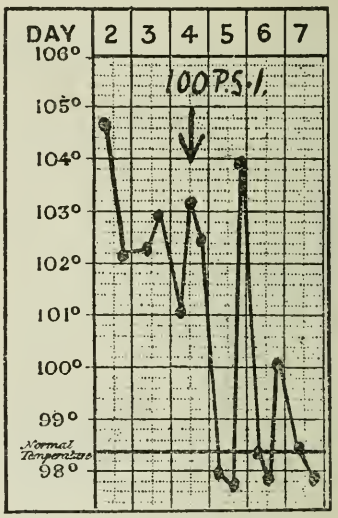
Case 7.—A man, aged 28. Extensive congestion of both lungs, with

patches of bronchial breathing, cyanosis, much distress. Injected on fourth day of disease with 100 millions streptococci and pneumococci and 60 millions influenza bacilli. Temperature fell rapidly and reached normal in 24 hours.

CASE 8.—A man of 19. Temperature, 104·6; highest pulse rate, 126; respirations, 28. Cyanosis and dyspnœa, with troublesome cough. Sputum abundant, frothy and pink. Considerable congestion at both bases, with



CASE 7.—*Influenzal Broncho-pneumonia. Male. Aged* 28.



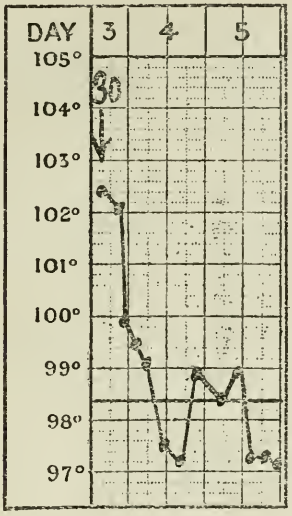
CASE 8.—*Influenzal Broncho-pneumonia. Male. Aged* 19.

patchy consolidation specially at right base. One hundred millions vaccine injected on the fourth day of the illness was followed by a fall of temperature to normal. The next day there was a rapid swing of temperature to 104° and back, and then temperature, after another swing to 100°·4, became normal. After the vaccine the respirations dropped from 28 to 20, and remained at that rate for the next few days. General condition rapidly improved.

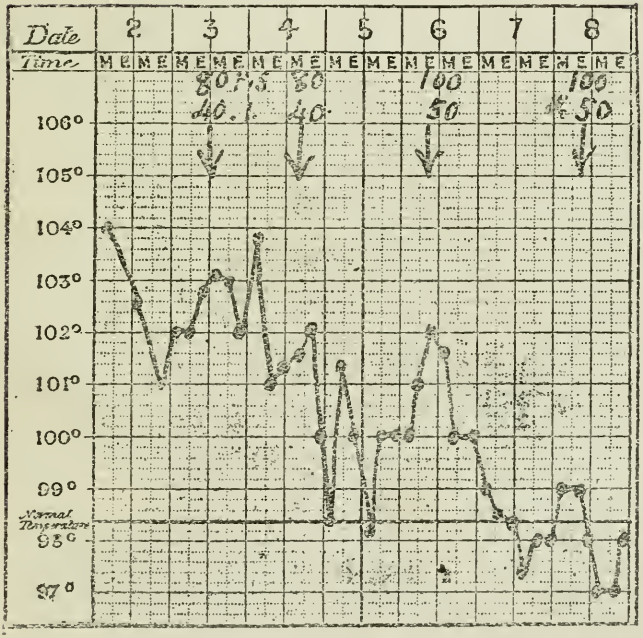
CASE 9.—A girl of 6, admitted on third day with broncho-pneumonia; temperature of 102°·2, respirations 48, pulse 152. An injection given immediately brought about a crisis followed by rapid improvement.

VERY SEVERE CONFLUENT BRONCHO-PNEUMONIA.

CASE 10.—A man, aged 50. Pronounced pulmonary symptoms from the beginning. A bad alcoholic history, with attacks of delirium tremens; stout and flabby, enlarged liver, albuminuria. Active struggling delirium during early stages, followed by stupor, muttering and picking at bedclothes



CASE 9.—*Influenzal Broncho-pneumonia. Female. Aged 6.*



CASE 10.—*Very severe Broncho-pneumonia. Male. Aged 50.*

and retention of urine. Very acute congestion of bases, followed by large confluent patches of pneumonia until whole of both lower lobes were solid, with many crepitations elsewhere. Very exhausting cough, but little sputum. Feeble pulse from the beginning, intense sleeplessness. One of the worst types in a bad subject Highest respiration rate 54; highest pulse rate, 108. The first injection of 80 millions streptococci and pneumococci, 40 millions influenza bacilli were given on the third day; the second of the same size on the fourth day. On the sixth and eighth days 100 millions pneumococci and streptococci and 50 millions influenza bacilli were injected, and three more injections were given after the temperature had reached normal. During convalescence left femoral thrombosis occurred. The chart shows that each inoculation was followed by a fall of temperature.

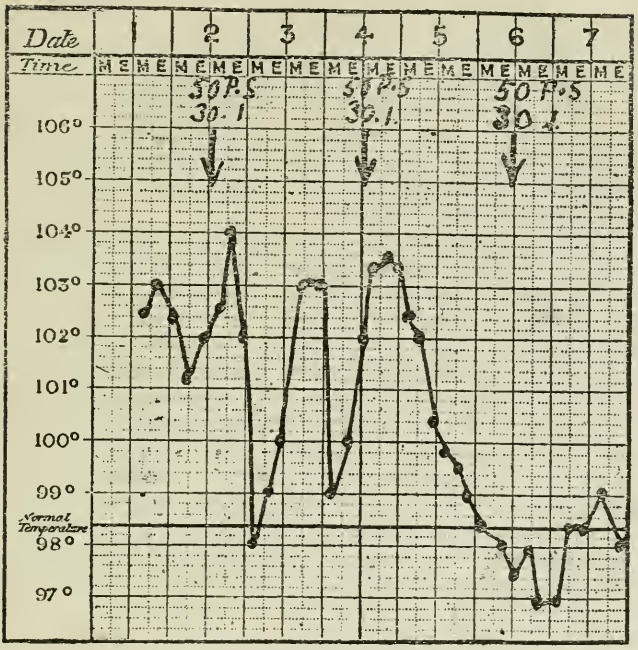
Case 11 in a woman of 26 was very similar. Onset was very severe, with early cyanosis, intense persistent headache and sleeplessness, large confluent areas of broncho-pneumonia in both lungs, frothy bloodstained sputum. Respiration rate was low—20 to 28 during the period of pyrexia—but rose to 48 after temperature had fallen and improvement set in. First dose of 50 millions of pneumococci and streptococci and 30 millions influenza bacilli was given on second day. Temperature fell by crisis within a few hours to 97, but rose again; a second injection of the same size was followed by a more gradual fall to normal. A third injection was given after temperature had reached normal.

Case 12.—A man of 28, was not seen until late in the illness. He was then apparently dying; stimulants and oxygen had been given for several days. Both lower lobes were nearly solid with large confluent patches, many crepitations throughout the rest of the lungs, pulse very feeble. After much hesitation, a dose of 80 millions streptococci and pneumococci, 50 millions influenza bacilli, was injected. This was followed by a gradual fall of temperature, accompanied by general improvement and a slow convalescence.

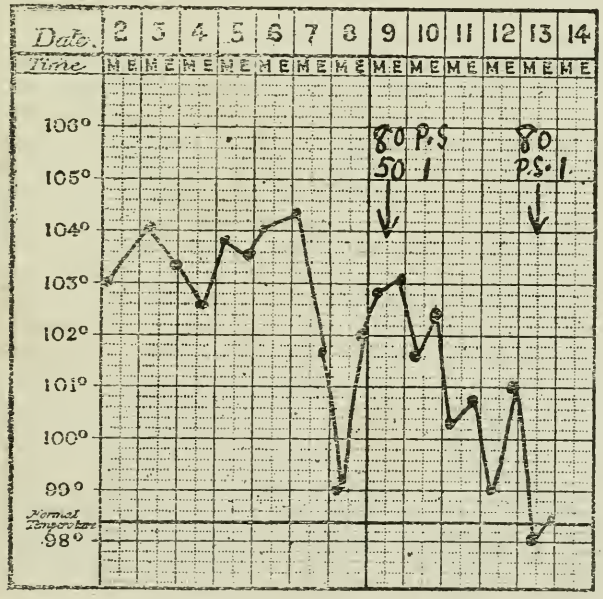
These cases gave rise to extreme anxiety throughout, and I feel confident that their lives were saved by the timely injection of vaccine bringing about an immunizing response.

The striking feature with the use of vaccines has been the almost constant fall in temperature following the injection. The earlier the injection is made, the more likely is the fall to be final. In cases injected after the second day, more than one injection is usually required before the temperature finally settles. Occasionally the first dose fails to cause a drop in the temperature, and a second larger dose is required to bring this about. If the fall of temperature occurred alone, or was followed by collapse or other bad signs, it would not be desirable, but the fall is associated with improvement in the aspect and in the pulse and respiration rate, which indicate that it is due to a definite immunizing response.

The youngest patient injected was a baby twelve months' old. She had broncho-pneumonia ushered in with convulsions, and was injected on the third day with 15 millions streptococci, pneumococci and influenza bacilli. When seen again 36 hours or so later, she was sitting up in her cot playing. The oldest patients were a man and wife, 69 and 68 years old, injected on the second and first day respectively. Both had much basal congestion of acute onset, and responded well. Children answer to vaccine treatment particularly



CASE 11.—*Very severe broncho-pneumonia. Female. Aged 26.*



CASE 12.—*Very severe broncho-pneumonia. Male. Aged 28.*

well, and take relatively large doses.

The important points for successful treatment are to give an adequate dose as early as possible after the onset of symptoms. Inadequate dosage and delay will cause disappointment. These points which I urged in an article on the Early Administration of Vaccine in Pneumonia,[2] written in 1914, have been amply confirmed by my experience since then. Vaccine can do little when the stage of cardiac and nervous exhaustion is reached.

The vaccine used has contained several strains of pneumococci, streptococci, and B. influenzæ. In a few cases, B. Friedlander has been added, because the sputum in these patients contained this organism. In some localities it might be desirable to add M. catarrhalis. Until the bacteriology of the disease has been more thoroughly worked out, vaccine treatment must necessarily be largely empirical, but experience has shown that these three organisms are most commonly associated with influenzal broncho-pneumonia, and a vaccine composed of them gives good results. The information so far collected points to the influenza bacillus being the cause of the cyclical epidemics, and having the power of enhancing the virulence of the organisms with which it is associated. It is the Bolshevik agent stirring up strife in many lands among a bacterial population of itself inclined to local outbreaks of violence. These organisms have been used in varying doses. My present plan is to give them in equal numbers. As the cases cited show, the doses have varied in size.

For an adult man I now recommend a dose of 80 to 100 millions of each organism; for an adult woman, 60 to 80 millions; for a child of 10 or 12, 30 to 50 millions; for a child of 2 or 3, 10 to 20 millions.

Space will not permit of a discussion of the theoretical points concerned in the use of vaccines for acute diseases. Experience teaches that reactions are much more easily produced in patients suffering from chronic localized infections, and that a negative phase is not to be feared in acute infections with doses of the size given above. The mode of action of a vaccine in acute diseases cannot at present be explained, but it is apparent that the patient's tissues can more easily utilize as antigen the dead germs contained in the vaccine than the living and rapidly multiplying germs within the body. During the incubation period there is rapid increase of the infecting organisms but no immunizing response, otherwise the infection would not develop. It is not until comparatively late in the disease that the manufacture of antibodies is sufficient to cope with the infection. Possibly one explanation is that the infecting germs surround themselves with " aggressins " which inhibit the immunizing machinery, whilst the injected dead germs are free from aggressins and so can be used as antigen. This absence of antibodies during the early stages and incubation period also explains the safety with which comparatively large doses of vaccine can be given. During the later stages antigen is provided in abundance naturally, and the immunizing machinery is stimulated to its utmost. Vaccine is then probably useless. Still

later, in conditions of delayed resolution or chronicity, vaccines again become useful.

SYMPTOMATIC TREATMENT.

The relief of *pain*, *cough*, and *sleeplessness* will indirectly assist in the production of immunity. Leeches, when obtainable, give quicker relief to pleuritic pain than any other measure. Light linseed and mustard poultices, applied to the congested areas in such a way as to avoid embarrassment of breathing, do much good. Dry-cupping might be revived with advantage. Efficient counter-irritation is far more important in these cases than in ordinary lobar pneumonia. Cough is often allayed by these local measures. Chloretone in liquid paraffin, 1 in 50, used with an atomizer for the throat and nose, may relieve pharyngeal irritability. Linctuses, containing codein, heroin, or tr. camph. co., may be used in the early stages, but are apt to derange the stomach and must be used sparingly. The cough, though troublesome, is necessary. Paraldehyde, dr. 1 or 2, in str onglysweetened tea, is the safest hypnotic. A combination of ½ to 1 dr. paraldehyde with 10 grs. of trional is often effective but less safe. In the severe cases with great excitement and struggling, one-hundredth of a grain of hyoscine is the only remedy likely to have effect. My experience with it has been fortunate; but it is an uncertain drug, and others condemn its use.

I frequently see bronchitis kettles in use, but in a disease with much frothy watery sputum the effect of saturating the air with moisture cannot be good. When sputum is scanty and viscid, small doses of iodides or ammonium chloride may be useful; but when, as is more usual, there is much bronchial secretion, these drugs by increasing it embarrass the breathing. On the other hand, $\frac{1}{120}$ gr. of atropine occasionally injected with discretion may give great relief.

In these influenzal cases, *circulatory failure* from depression of the medullary centre and consequent vasomotor paralysis seems to play as important part as direct heart failure. When these are present, our difficulties are great. One great advantage of cutting short the disease in its early stages by vaccine treatment is that circulatory failure is prevented. Much may be done in the way of prevention by aerotherapy, the avoidance of exertion, the administration of cane sugar, malted milk, honey or milk sugar, to feed the heart, and the administration of oxygen. Many physicians regard the use of oxygen as disappointing, but no remedy is more badly administered. The futile method of holding a funnel over the patient's face is still commonly practised even in first-class hospitals. I have even seen an india-rubber tube fastened to the pillow, with the open end several inches away from and below the patient's face. Administered through a proper mask and warmed, I have no doubt of its good effects. An excellent method is to enclose the head in a hatbox with a piece cut out to fit the neck, and the oxygen tube passed through a hole in the side. It should be administered for five or ten minutes at a time.

For vaso-motor failure, *pituitrin* ½ cc. every six hours, and *camphor*

grs. 3, in ether min. 3 and olive oil min. 12, given every four hours, in both cases intramuscularly, have given me the best results. Five minims of liq. strychninæ injected four hourly, and alternating with the camphor injections, may be of help in vaso-motor failure. In spite of common opinion, experiments show that it has no action upon true heart failure. It is a powerful stimulant to the respiratory centre, but this centre is already over-stimulated by its natural excitant, carbon dioxide. With *digitalis* and *strophanthus*, we reach highly debatable ground. Their use has again risen in favour, but there is no experimental evidence to support the giving of digitalis to the heart in fever. It is a case of *tot homines, tot sententiæ*. For curiosity I looked up the references on the use of digitalis in fever in various sections of a recent encyclopædia of medical treatment, and found the following opinions: "Digitalis more often does harm than good"; "Digitalis is undoubtedly a useful drug" (in pneumonia); "To attempt to reduce the rate of the heart in fever by administration of cardiac glucosides is, of course, futile." I have used digitalis in many preparations and doses, early and late, but have never seen any result of value. To give to a poisoned heart a drug, which only acts by its poisoning effect, cannot but be bad.

Alcohol has also come into favour again, and in many cases seems to be the only drug used. Beyond the temporary reflex stimulation caused by the local effect upon the mouth, pharynx and stomach, it is now well established that alcohol is principally a depressant. It also lowers the resisting power to infection. Patients accustomed to alcohol are able to detoxicate the amounts usually given, and in such subjects it may be continued without harm. But to give, under the guise of a stimulant, a depressant drug which moreover inhibits the immunizing processes to patients who are unable to neutralize its effects, cannot be sound treatment. Still, the pharmacologist must give way to the practical experience of the physician if the latter can prove his case. But no proof has yet been advanced that alcohol is of service in circulatory failure.

Of other drugs, *caffein citrate* 5 grs., three times a day in a strong cup of coffee, frequently acts well. In collapse, *saline infusions* with *glucose* 5 per cent. injected subcutaneously or very slowly into a vein have a distinct life-saving value and are too seldom used.

Only the confirmed therapeutic optimist can face with equanimity these cases of failure from toxic exhaustion. It is difficult to refrain from giving some drug; but the truth must be faced, that we have no drug of undoubted value—no drug, even, of which the indications for its administration are clear. Our real hope lies in the establishment of sound lines of specific treatment, so that by its early administration broncho-pneumonia may be robbed of half its terrors.

REFERENCES.

[1] *Journal of Vaccine Therapy*, September, 1912. *Clinical Journal*, April 30, 1913

[2] *British Medical Journal*, March 13, 1915.

THE NERVOUS SYSTEM IN INFLUENZA.

By WILFRED HARRIS, M.D., F.R.C.P.

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I HAVE not much to add to my article, which appeared in THE PRACTITIONER in January, 1907. There is no acute malady after which disturbance of the nervous system is so frequent as after influenza, and none that has such varied nervous sequelæ. We may go even further than this, and scarcely be accused of exaggeration, if we assert that the prime sufferer in influenza is the nervous system, and that the large majority of its symptoms and sequelæ are the effects, direct or indirect, of the expenditure of the virus of Pfeiffer's bacillus upon nervous structures. Numerous and diverse as these may undoubtedly be, there is scarcely a disease of nerve cell or fibre that has not been ascribed to this most searching of diseases since the last great pandemic of the early nineties. We must, therefore, be on our guard against the facile error of debiting every *post hoc* as *propter hoc*, for, although it may be sometimes a justifiable euphemism to speak of a locomotor ataxy or general paralysis as influenzal in origin, we must not overlook the essentially syphilitic nature of these diseases, and allow ourselves as well as the friends of the patient to be the willing victims of this delusion. No doubt, in many cases, the first symptoms of these diseases have appeared within a comparatively short time of a severe attack of influenza, and it is quite easy to understand how this disease, which exercises so profound a depressant effect upon the nervous system, may aid the development of symptoms of diseases which were previously latent. Although, in this manner, influenza may contribute to the production of tabes or general paralysis, it is probable that they are never, strictly speaking, influenzal sequelæ, and dependent solely, in their ætiology, upon the antecedent febrile attack.

It will be convenient to describe the effects of influenza upon the nervous system in the following three groups:—(1) The nervous symptoms appearing at the onset; (2) complications arising during the illness; (3) sequelæ.

The nervous symptoms appearing at the onset are well illustrated in the account of his own attack that a London physician gave some time ago. He states that he awoke on Boxing Day, 1889, feeling severe pains at the back of the eyeballs, frontal headache, and pains all down his spine. The pains were symmetrical, and slightly shifting in character, affecting all the posterior muscles, beginning at the neck and ending at the calves. There was entire absence of pain in the anterior muscles of the trunk and limbs; he felt extreme prostration

and depression of spirits, accompanied by a feeling of nausea and pain in the epigastrium resembling sea-sickness. The severity of the symptoms lasted for two days, continued in less degree until the thirteenth day, when they disappeared completely.

After an incubation period of about two days, the onset of severe frontal headache, pains in the eyeballs, and in the back and limbs, with extreme prostration, are characteristic of the disease. Even before these acute symptoms appear, an unaccountable loss of muscular power may be noticed by the patient. A young man of 20, who was rowing hard daily in an eight-oared boat in training for the Lent races at Cambridge, in February, 1890, noticed for two days, though he was feeling otherwise quite well, that he was unable to pull his weight in the boat, and to work so hard as he was accustomed to do, when suddenly, about eight in the evening, he was attacked by severe cramping pains in the back and calves and by such excessive weakness that he was scarcely able to make his way back to his rooms and get to bed. This suddenness of the onset, of the collapse, and prostration in influenza is most striking, and Goodhart remarks that only in cholera is the collapse so sudden and continuous, flinging strong men down. Sudden faintness or syncope may be the first sign of the advent of the disease, as in the case of a man who was driving a trap along a country road, when in a moment he fainted and fell out of the trap on to the road. Recovering consciousness in a second or two, he was able to climb back to his seat and drive back home, where he was seen to be looking extremely ill and shivering, and was put to bed. He then went through a characteristic sharp attack of influenza, but could remember nothing about his drive from the time of starting.

The headache at the onset is nearly always frontal, and is often unusually severe, preventing all sleep, and racking the patient to such a degree with torture that he may become completely obsessed by the pain, and apparently delirious. The severity of this headache usually subsides after two or three days, though in some cases it is less noticeable at the onset of the illness, but may come on towards the end of the first week, when the fever and the muscular pains are beginning to subside. Frontal headache, accompanied by pyrexia, is not pathognomonic of influenza, since the two often usher in typhoid fever or small-pox, but, in addition to these symptoms, the sudden appearance of *severe pains in the back and limbs*, and especially at the back of the eyeballs, together make up a clinical picture that can scarcely be mistaken for anything else than influenza. The pains in the back-muscles, calves and thighs may be very severe and shooting, resembling the lightning pains of tabes, or they may be more general; the patient complaining of a feeling of soreness all over, as though he had been thrashed with sticks. Occasionally the pains appear to be situated in the joints, and may be mistaken for rheumatism, or they may seem to be in the bones, or in the chest, lumbar region, or in the testis. The neuralgic pains about the thoracic or abdominal viscera may simulate various disorders of these organs, and may be mistaken for pleurisy, pericarditis, stone in the

kidney, or bladder, hepatic or intestinal colic; these severe pains rarely last more than two or three days. The onset of the acute symptoms is almost always accompanied by a certain amount of pyrexia, the fever usually ranging from 101° to 104°, though all variations may be met with; temperatures of 105° are not very uncommon, and even hyperpyrexia may occur. It is undoubtedly true that a certain number of cases of influenza may show no pyrexia at all, although the other symptoms may be more or less characteristic of the disease. It has often been remarked that the rate of the heart-beat in influenza is disproportionately slow to the fever that may be present, a pulse of 80 or 90 being not uncommon with a temperature of 101° to 102°. The tongue is usually large and indented, and soon becomes covered with a thick creamy fur, while the breath becomes peculiarly fœtid. *Sweats*, too, often severe and drenching, are not uncommon during the course of the fever, and it has been remarked that the perspiration, like the breath, has a peculiar sickly odour. With the sweats, *rigors* are likely to occur, and a shivering fit may even mark the onset of the disease. A peculiar feeling of giddiness and light-headedness may be a most distressing early symptom, and is likely to be associated with an intense feeling of mal-de-mer, retching, and vomiting. The vomiting may resemble sea-sickness in its noisy, spasmodic character, and may be much intensified by food.

In some attacks, repeated fits of sneezing, with the onset of severe coryza and running at the eyes, may usher in the illness. The sneezing and running at the nose and eyes may resemble hay-asthma, while, in some patients, there may be spasmodic attacks of wheezing and dyspnœa resembling ordinary spasmodic asthma, with great respiratory distress and lividity of the face. The precordial sensation of oppression may be so distressing as to resemble angina pectoris, and the patient may complain of a feeling as if the heart would stop at any moment. Suddenly as the disease may make its appearance, its course is apt to be comparatively protracted. The severity of the pains in the back and limbs, the fever, and the frontal headache, are usually much diminished after two or three days, and in the slighter cases these symptoms may completely disappear in this time. A most characteristic symptom of influenza is, however, likely to be left behind, an extraordinary muscular weakness and depression of spirits, which frequently last from one to two weeks. In the favourable cases, which form the majority, there may be no further complications ; the intense lassitude and depression clear up in a few days, and the illness is then over. In a great number of instances, however, the story is not so simple, and the tale of symptoms is lengthened by a formidable list of complications and sequelæ that may arise in connection with the disease.

Complications.—In addition to the slow heart and fainting, with sudden syncopal attacks that may occur at the onset, the action of the heart may later become irregular or extremely rapid. A permanent degree of dilatation of the heart with irritability of action

and tendency to tachycardia may follow this condition, and even Graves's disease with exophthalmos and goitre may ultimately develop. Other vascular lesions in connection with the nervous system are cerebral thromboses. Right hemiplegia, with motor aphasia and agraphia, has been recorded, due usually to thrombosis of the middle cerebral artery, but *polioencephalitis* may be the cause, the general symptoms suggesting meningitis, with presence of Kernig's sign, though the cerebro-spinal fluid obtained by lumbar puncture shows neither increase of cells nor organisms present. Sudden onset of blindness has also been observed, clearing up later and leaving permanent right hemianopia, due to a vascular lesion in the occipital region, probably a thrombosis, causing a patch of softening in the calcarine cortex on the left side. The headache may fail to improve after the first two or three days, becoming worse with the development of symptoms of *meningitis*. The pain in the head then shifts somewhat in position, becoming chiefly occipital and vertical. The pain is apt to spread down to the back of the neck, which becomes stiffer and more tender, with perhaps slight head retraction. The optic discs become blurred and swollen, and there may be also some symptoms of spinal meningitis, with severe pains in the limbs and tenderness along the nerve trunks, drawing up of the legs, and increased or absent deep reflexes. The cranial nerves as a rule escape, with the exception of convergent strabismus from sixth nerve weakness, and some difficulty in swallowing from pharyngeal and palatal palsy.

Amongst the most important of the complications are the *ocular phenomena*. *Optic neuritis*, with choked discs and retinitis, was not unfrequently seen in the epidemic of the early nineties. It is apparently a toxæmic symptom, and does not indicate the presence of meningitis, in which condition the papillitis rarely amounts to more than blurring of the edges of the disc, with swelling of one or two dioptres. *Influenzal retinitis* is peculiar from the frequent presence of starry, fan-shaped groups of white spots in the region of the macula, and between the macula and the optic disc. Vision may be seriously damaged by *retro-bulbar neuritis*, due to a hæmorrhagic inflammation of the macular bundle of fibres in the centre of the optic nerve, producing central scotoma with great diminution of acuity of vision and loss of discrimination of colours. Embolism and thrombosis of the central artery of the retina have also been recorded, causing sudden blindness, or great defect in sight if only a branch of the artery is occluded. Irido-choroiditis, with its consequent injury to vision through occlusion of the pupil, vitreous opacities, and adhesions of the iris to the lens capsule may also be serious. Comparatively common were varying degrees of *ophthalmoplegia*. The most frequent of these is loss of accommodation, due, no doubt, as in post-diphtheritic neuritis, to a localized paralysis of the ciliary muscle. Mostly the pupils, too, become sluggish in their reaction to light when there is loss of accommodation, and in a very few cases the reaction to light is completely lost. In addition, there may be ptosis, paralysis of any of the external

ocular muscles, or even complete third nerve palsy. Goldflam recorded a case which developed complete bilateral ptosis, with total immobility of both globes, following 14 days after influenza. The pupils were normal, but there was weakness of the soft palate with difficulty in swallowing. Gillet de Grandmont published a somewhat similar case with bilateral ptosis and complete external ophthalmoplegia, with weakness of accommodation and sluggish pupils. Other severe symptoms present were double optic neuritis and pronounced somnolence, with weakness of the extremities. Oppenheim similarly has observed double ptosis and bilateral ophthalmoplegia, with weakness of accommodation following influenza. In addition there was difficulty in articulation and in swallowing, with slight fever, severe tachycardia, and paresis of the extremities. This case recovered in five weeks.

Gutmann recorded a case in which there developed early a complete third nerve palsy, with the eyeball turned outwards and motionless, and complete ptosis; the pupil was dilated and inactive to light. Later on the external rectus also became paralysed, and the superior oblique as well, the eyeball being fixed. Ultimately the ocular paralysis cleared up, and the reaction of the pupil to convergence and accommodation returned, though it remained inactive to light. These examples of severe opthalmoplegias complicating influenza are somewhat rare, but the slighter forms of paralysis of single muscles, and especially of loss of accommodation, were fairly common during the epidemic. It will thus be seen that the ocular paralyses which occur in influenza last longer, and are more severe than the post-diphtheritic forms of ocular paralyses. I can find no record of any autopsies on these cases, and Wilbrand suggests that they are due to a hæmorrhagic polioencephalitis, affecting the region of the third, fourth, and sixth cranial nuclei in the pons, *polioencephalitis inferior*. Leichtenstern first described encephalitis as a special complication in influenza, and Goldflam has recorded a case of polioencephalitis, superior and inferior, with acute anterior poliomyelitis, followed by death, after influenza; that is to say, an acute hæmorrhagic lesion of the grey matter of the cortex, of the pons, medulla and of the anterior horn cells in the spinal cord, occurring at the same time as a fatal complication of influenza. *Acute encephalitis*, or polioencephalitis superior, is usually ushered in with a convulsion, which may be unilateral; or in children, with a series of convulsions, followed by prolonged coma lasting perhaps several days. When occurring as a complication of influenza, it is likely to occur between the second and seventh days of the illness. It is a toxæmic infection of the meninges and pial vessels with Pfeiffer's bacillus of influenza, and is likely to be accompanied by signs of meningitis also. The grey matter will be found to contain minute points of hæmorrhage, and the nerve cells swollen and exhibiting signs of chromatolysis. Lumbar puncture in these cases may be successful as a means of demonstrating influenza bacilli in the cerebrospinal fluid withdrawn. If the case recovers, the symptoms, resulting

from the acute cerebral lesion, will depend upon the part of the cortex that has suffered. If the Rolandic area has borne the brunt of the attack, hemiplegia will result; if the frontal lobes have been the chief site of the mischief, then permanent mental impairment will ensue; if the occipital cortex has been attacked, then impairment of vision will be the result, at first probably complete blindness, which may be in rare cases permanent and complete, but if one occipital lobe has suffered more severely than the other, then the visual field is likely to clear up on one side, leaving behind a permanent hemianopia; sometimes the whole visual field may recover, as I have seen after complete blindness lasting for two weeks. Further evidence of the poisoning of the cerebral centres by the influenzal toxin is afforded by the peculiar dazed feeling and apathy, with extreme depression and bad dreams, that often occur as initial phenomena in the disease.

Hysteroid *convulsions* may first draw attention to the patient's serious condition, and they may be followed by fainting, stupor, or even a cataleptic state. Grasset and Ranzier record the case of a French soldier, æt. 28, who had a hysteroid fit on the second day of his illness, the arms and legs being acutely flexed, lasting half an hour without loss of consciousness, but involuntary urination afterwards. This was followed by temporary right hemianæsthesia, which after a short time cleared off except from the arm. He was completely unable to move the wrist or fingers, and there was complete analgesia of the hand up to a circular line above the wrist, though tactile sensation was unimpaired. There was also loss of the pharyngeal reflex. As an instance of *catalepsy* the following case may be cited :—A man was seized with influenza, with shivering, abdominal pains, vague pains all over, constipation, anorexia, restlessness, and temperature 103°. After four days the symptoms subsided sufficiently for him to be able to get up. He then suddenly lost consciousness, and was found lying on the bed apparently senseless, with his eyes open, the face pale, and pulse weak, 120 per minute. There was generalized tonic rigidity of the muscles, so that the limbs would remain in any position in which they were placed, so-called lead-pipe rigidity; this state lasted for two hours, and was followed by recovery. At the time of the epidemic in 1889–90, there was considerable scare in Switzerland over the reported appearance of "*Nona*," or a condition of apparent death. It is a condition of extreme somnolence, or deep trance, simulating death, but it is not peculiar to influenza, and has been seen in cerebro-spinal meningitis. A case of apparent death of a man was reported in 1890. After due consultation a certificate of death was signed; an hour before the time fixed for the funeral, signs of recovering consciousness were noticed by the friends, and, as the result of efforts to restore animation, he recovered to the point of recognizing those around him, but he again lost consciousness and died.

The following case of *trance* after an attack of influenza was reported by Dr. Nathan Raw. A woman, always hysterical, and a deep student of the Bible, had a severe attack of influenza, with excruciating pain

in the head, and was quite prostrate in mind and body for two weeks. She then fell into a deep sleep, and dreamt that she heard a voice saying to her, "You are dead." She lay quite helpless and flaccid for ten days, and could not be roused by any means, nourishment being given by the stomach tube; she afterwards recovered rapidly. Young infants, if seized in the grip of influenza, are more likely to be overcome by the toxæmia, and lie in a somnolent state. This may be compared with the different way young infants react to acute lobar pneumonia as compared with adults. Instead of the active delirium and sthenic symptoms exhibited by the latter, the babe lies in a drowsy condition as though drugged. So, in influenza, they may lie in a prolonged somnolent condition throughout the febrile attack for a week or ten days, and then recover rapidly.

Certain visceral symptoms, occurring early in the course of influenza, may be attributed to disturbance of the nerve supply to these organs by the infection. Such are the neurotic polyuria and the watery diarrhœa that are sometimes met with. Constipation is, however, more frequently present. Spinal disorders, that may complicate influenza, are acute anterior poliomyelitis, disseminated myelitis, ascending myelitis, Landry's paralysis, and herpes zoster. Spastic paraplegia has also been recorded as a sequel, but unless it is the result of an acute attack of myelitis occurring as a complication, it is doubtful if the spastic paralysis can rightly be included as a direct result of influenza. Jolly saw a case of poliomyelitis in a lady, which came on three weeks after influenza with pains and paralysis of the left arm, and later of the left leg. Fiessinger records a case of polioencephalitis inferior in a young girl of 18, who, after the usual symptoms of influenza, developed, on the fifteenth day, nasal speech and difficulty of swallowing, with opisthotonos. The motor and sensory power of the extremities was good; pulse 160, followed by death; no autopsy. Bulbar crises, resembling the attacks seen in children with diphtheria, with spasm of diaphragm or glottis and inability to swallow, have been met with, and may be rapidly fatal.

Myelitis, complicating influenza, is usually cervical and accompanied by meningitis, or it may be scattered in separate foci over a large area of the spinal cord. Paraplegia is the prominent symptom, at first flaccid, and, if the patient survives long enough, spasticity develops later. Incontinence of urine and fæces, anæsthesia of the lower part of the trunk and lower limbs, sometimes muscular wasting in groups of muscles distributed according to spinal segments and not to their peripheral nerve supply, and rapid onset of severe bedsores indicate a severity of the spinal injury from which recovery rarely occurs. In one case high dorsal myelitis was followed a week later by severe failure of vision due to large central scotomata from double retrobulbar neuritis. Idiopathic tetanus has more than once been recorded as following a few days after an attack of influenza, but precisely in what way the influenzal attack assists the advent of the tetanus bacillus to the nervous system, we are completely ignorant. Féréol, amongst others, has recorded a case of *ascending myelitis* com-

plicating influenza. This is invariably fatal, starting with loss of power in the lower extremities and anæsthesia of the gluteal region, spreading up the legs and trunk, so that by the end of a week the whole of the lower portion of the body below the arms may be completely anæsthetic and powerless, respiration being carried on entirely by the diaphragm. The reflexes, superficial and deep, are lost from the beginning, and if the patient survives for more than a week, reaction of degeneration in the muscles of the lower extremities will be found on electrical testing.

Landry's paralysis, so called, may also occur, differing from ascending myelitis clinically in the absence of any pronounced degree of anæsthesia or muscular wasting, and little or no involvement of the sphincters. In their morbid anatomy, however, there is a certain resemblance, though occasionally a case of Landry's paralysis may survive. In cases of ascending myelitis, the whole cord, more especially in the sacral and lumbar region will be found swollen, more or less diffluent on section, with small hæmorrhages scattered throughout both grey and white matter. The pia-arachnoid covering the cord is also involved in the inflammatory process, and the condition is really a spreading meningo-myelitis. In many of the fatal cases of Landry's paralysis, which have come to autopsy, either nothing abnormal has been found, or slight changes of a chromatolytic nature of the nerve cells and some swelling of the fibres. In some others, though the cord and pia-arachnoid showed very little change, the dura mater covering the cord was found inflamed, and the seat of a micrococcal infection, which suggested that, in some cases at any rate of Landry's paralysis, the spinal theca is the part specially attacked.

Whether, in the cases of this affection complicating influenza, Pfeiffer's bacillus may similarly invade the spinal dura and produce these symptoms, we have no knowledge; in all probability, the bacillus of influenza is capable of producing a spreading meningo-myelitis, which may be either slight or severe, the less severe forms having been described clinically as cases of Landry's paralysis complicating influenza.

Herpes zoster, as is only to be expected, has frequently occurred during the course of influenza. Zoster has been shown to be an acute infection, resembling in character acute anterior poliomyelitis. In the latter, it is the anterior horns that suffer almost exclusively, while in zoster the stress is borne by the posterior root ganglia, in which hæmorrhages and acute degeneration and swelling of the nerve cells have been found. Most commonly it attacks one of the dorsal roots, so that pain and redness of the skin round one side along the course of an intercostal nerve is the first symptom, followed by a crop of vesicles mapping out the distribution of the posterior root which is attacked. When, as is usually the case, it is one of the dorsal roots that suffers, the vesicles are found in two positions especially, behind, over the posterior division of the nerve, and in front, for four or five inches along the intercostal space up to the middle line. Zoster leaves permanent scars, but unless the patient attacked by it is over sixty, it leaves

no other permanent effects. In old people, however, it may be followed by the most persistent and intolerable neuralgia, which nothing short of division of the posterior root internal to the ganglion will cure. Patients have indeed been driven to suicide by the unbearable severity of the pain. Zoster has been met with in influenza over the the neck and occiput, affecting the higher cervical posterior roots, and this has been accompanied by facial paralysis on the same side. The Gasserian ganglion being precisely comparable anatomically with the spinal posterior roots, it is not surprising that herpes zoster sometimes occurs over the face and forehead along the distribution of the fifth nerve. Herpes frontalis, or ophthalmicus, is a special form which attacks the first division of the fifth nerve only, and may produce serious ulceration of the cornea in addition to the outcrop of vesicles over the forehead and side of the nose.

In addition to the cerebral and spinal diseases already referred to, which may arise in the course of an attack of influenza, the peripheral nerves also may suffer, and severely. These may be affected either as a local neuritis, or as a peripheral multiple neuritis, or by neuralgic pain only.

Local neuritis may occasionally attack certain cranial nerves, such as the fifth or seventh, causing weakness of the jaw muscles with pain and anæsthesia over the face, forehead, tongue, and inside of the cheek, when it is the trigeminal that suffers, and paralysis of the facial muscles or Bell's palsy, when the seventh is damaged. The eighth cranial nerves have also been known to suffer in influenza, causing a peripheral nerve deafness. This is to be distinguished from the deafness that may result from the middle ear disease and mastoid trouble, which follow an initial abscess in the ear, with severe pain, that not uncommonly is an initial symptom in an attack of influenza. The ocular paralyses have already been dealt with, some of which, especially the isolated muscular palsies of external or internal recti, or loss of accommodation, are due to neuritis of branches of the third nerve or the abducens, while the more severe cases of total ophthalmoplegia are much more probably due to a hæmorrhagic lesion in the floor of the aqueduct, known as polioencephalitis inferior.

Neuritis of the brachial or lumbar plexus on one side producing brachial or sciatic neuritis has been fairly common after influenza.

Brachial neuritis develops with rapid onset of intense pains down one arm from shoulder to fingers, made worse by the slightest movement, and entirely preventing sleep. It is due to a perineuritic inflammation of the sheaths of the nerves entering the plexus, a condition which also may occur in gouty persons, or from rheumatism, or exposure to cold. In a severe case, the arm becomes swollen, œdema appearing on the hand and wrist; the skin becomes shiny, and ultimately the muscles waste and trophic lesions appear on the skin and nails, glossy skin, wasted finger pads, and stiffness of the joints, especially of the fingers and wrist, from periarticular adhesions. In less severe cases there may be complete recovery after about a month.

Sciatic neuritis, or sciatica, is a similar process affecting part of

the lumbar plexus, causing severe pain down the back of the thigh and in the leg and foot. The chief site of the pain is often in the ankle, with tenderness along the sciatic nerve on pressure in the mid-gluteal region, back of thigh, or popliteal space. With severe attacks, swelling of the foot and ankle and even the appearance of bullæ on the skin are found. Movement, as in brachial neuritis, intensifies the pain, and a severe attack may similarly be followed by trophic lesions of skin and muscles, though this is, on the whole, a more uncommon result in the leg than in the arm.

There is scarcely a nerve twig, or nerve trunk, that has not been known to suffer from influenza, with resulting local tenderness and trophic symptoms of skin or muscle persisting for variable periods. As was said in the case of herpes zoster, so with the innumerable post-influenzal nerve inflammations; in young people, the trouble generally clears up entirely, leaving behind no permanent ache nor pain, while in older persons, roughly in proportion to their weight of years, influenzal neuritis is more difficult to get rid of, and frequently leaves behind most intractable neuralgia and tender points, even though there may be no more definite signs of trophic disorder of muscle, skin, or joints.

Peripheral Multiple Neuritis when severe is one of the most formidable complications that occur in the course of influenza. It is one of the later complications to appear, generally three weeks after the onset of the illness, but it may be as long as six weeks. It is really a complication rather than a sequel, as the morbid process is a toxæmic parenchymatous degeneration of the nerve fibres themselves, thus differing from the perineuritic inflammation of the sheath of single nerves or nerve plexuses, which is found in brachial neuritis, or sciatica, or other forms of local neuritis. The multiple neuritis occurring after influenza is similar in character to that which occurs after other toxæmic infections, such as diphtheria, varicella, measles, pneumonia, typhoid, beri-beri, septicæmia, and other infections of microbic origin, or the multiple neuritis that follows chronic poisoning with alcohol or arsenic. It is, therefore, unnecessary to describe the form of the neuritis at length, but to mention its chief features and the points of difference, which will help to distinguish it clinically from the multiple neuritis due to these other toxic causes. Considerable pain may mark its onset, with shooting pains in the limbs, and general nerve tenderness, and weakness of the extremities, especially of the feet and ankles. The form of the paralysis is rather more motor than sensory, and muscular wasting with reaction of degeneration may ensue. Paralysis of the lower extremities, loosely described as paraplegia, is probably in most cases due to multiple neuritis. In a few cases, dangerous complications from cardiac and respiratory paralysis may be met with, but they are far less common than in diphtheria or beri-beri, while the sensory changes, anæsthesia, etc., are usually much less marked than in the alcoholic form. Bilateral facial paralysis may occur as part of a multiple neuritis due to influenza, as it may from alcohol, or the rare form of rheumatic general multiple neuritis. As in other forms of multiple neuritis, there is no sphincter involvement unless there is, at the same

time, a patch of myelitis developed. Influenzal multiple neuritis is much less likely than diphtheria to begin with cranial nerve paralyses, though the ocular and other cranial nerve paralyses, when they do occur, are more severe than in diphtheria, but are due to local neuritis or a hæmorrhagic polioencephalitis, and are not a part of the multiple neuritis.

Peripheral neuritis in influenza, though a severe, is rarely a fatal complication, and requires treatment on the usual lines of multiple neuritis, with rest in bed so long as there is any sign of the neuritis increasing, and later massage, exercises, strychnine, and galvanic treatment.

Neuralgia has already been mentioned as occurring after herpes zoster, or local neuritis of single nerves or of a plexus. Trigeminal neuralgia may date from an attack of influenza in which there were at the time no signs of actual neuritis of the fifth nerve. Neuralgia differs anatomically from neuritis in the absence of any discoverable lesion in nerve-cell or fibre in the former, but the term is often used loosely, by expert physicians as well as by the laity, for the severe lancinating pains radiating along the course of a nerve trunk and its branches, which may be reflex pains from irritation of a single twig, as in a carious tooth, or the after pains left behind after a severe neuritis, as in herpes zoster, brachial neuritis, or sciatica. The treatment of these neuralgic pains must be attempted on general lines, though perhaps post-influenzal neuralgia, more than other forms, may be controlled by quinine. When post-influenzal supra-orbital neuralgia persists in spite of such treatment, alcohol should be injected into the nerve. The same treatment of trigeminal neuralgia of the second or third divisions of the nerve will give immediate and lasting relief.

Many SEQUELÆ have followed influenza, some of which, more than others, are directly dependent upon the disease. One which is perhaps peculiar to influenza is the prolonged *loss of power of smell and taste*, which often lasts for many months, or a year, after the acute illness. It is usually the sense of smell alone that is really affected, the power of appreciating salt, sweet and bitter, being retained, but all flavours and the taste of food are not distinguished on account of the loss of smell, which is probably due to a neuritis of the olfactory nerves. I have known it permanently lost after influenza.

Progressive bulbar palsy has also been recorded, with paralysis of the vocal cords, palate and pharynx, and tremors of the lips and eyelids, beginning acutely a few weeks after influenza, and steadily progressing.

Myasthenia gravis, too, has been recorded by Remak, in a girl of twelve, following five months after influenza, beginning with bulbar symptoms and ending with attacks of intense dyspnœa, death occurring in four months.

Epilepsy has been noticed to recur during an attack of influenza after years of intermission, and to continue in an aggravated form. This is probably due to a certain degree of cortical damage from

encephalitis, for epilepsy is a common sequel to this dangerous cortical disease.

Migraine, too, may become aggravated in frequency and intensity.

Parœsthesiæ of all sorts and descriptions have been described as sequels, including pins and needles sensation, vague pains, numbness, hyperæsthesia, etc.

Infective polyarthritis and *diabetes*, too, may be formidable legacies of influenza. The polyarthritis, though not a nervous complication, may be mistaken for paraplegia, or for multiple neuritis, with its subsequent fixation of joints from periarticular adhesions.

Lastly, we may mention the extraordinary effect that influenza often produces upon the mental functions. *Loss of memory*, and an intense *inertia of mind and body* may last for weeks and months after the acute illness, and all the well-known symptoms of grave neurasthenia, as well as more grave mental disorders, may remain and become permanent. This is in all probability due to the severe toxæmia from which the cerebral cortex has suffered during the infection; this may not in all cases produce lesions of nerve cells and the grey matter, that can be recognized as due to an encephalitis, yet the function of this grey matter may be permanently damaged by the poisoning, though modern methods of staining are unable to recognize the changes. This condition is in fact similar to the grave neurasthenia that may result after any severe mental shock, such as the complete upset of nervous balance that has been observed so frequently after prolonged battle strain or burial from shell explosion. As with these latter, it is almost invariably those with a neuropathic family history that suffer most.

Practical Notes.

THE PRESENT EPIDEMIC OF INFLUENZA.

This curious disease, which was so mild in March, 1918, occurring as a three-day fever very similar to dengue, has become very much more serious and at times as fulminant as certain outbreaks of plague or cholera in August and September, 1918. It is the same disease that was spoken of in 1889–90 as infectious *grippe* and influenza, as may be seen by referring to the literature of that time. It is a most infectious disease, as it was in the epidemic of 1889. A mother spends an hour at the bedside of her suffering son, falls sick next day, and is dead three days later. It spreads very rapidly, particularly in all crowded centres, such as barracks, depôts, seaport towns, schools, and camps of instruction.

The disease attacks all ages from infancy to old age, and is specially serious for old and debilitated subjects, but at the same time robust young men, like recent conscripts, are frequent victims.

It varies greatly in severity, proving in some places fatal to about 50 per cent. of cases, and in others being very mild. It became serious during June in Spain and Switzerland, and in France in August and September. It confers no immunity, re-infection is by no means rare, and relapses are common.

In the *mild form* of attack, the patient shivers quite suddenly or feels out of sorts, and has to take to his bed within half-an-hour after appearing to be in perfect health. He has a slight headache and complains of smarting in the throat. He becomes depressed, heavy, and feverish, with his eyes bright and slightly injected. The temperature will be found to be 102° to 104°. On the following day, a coryza has appeared, and there is a raucous cough with no expectoration. The temperature is 104°, and the pulse 100. In some cases, the rate of the pulse is not increased in proportion to the rise in temperature, as is observed in typhoid fever. The cough is very occasionally painful, and a very small quantity of sputum is expectorated with much difficulty. A few sibilant râles may be heard during the coughing fits. The tongue is coated, and there is complete loss of appetite.

After three or four days, the temperature falls to 98°·6, but the headache gets worse, and pains are felt in the limbs, the patient feeling very tired. The depression and tired-out feeling last for a week or two. Relapses are not uncommon on the fourth day of apyrexia. They are always more serious than the first attack, all the symptoms are exacerbated, and convalescence is prolonged.

In the *fulminant form*, death occurs in from 24 to 48 hours, after a sudden onset accompanied by suffocating dyspnœa. The patients become cyanosed in a few hours, and the pulse cannot be counted.

The *acute forms* comprise the respiratory forms especially: pneumonia, with a large area of hepatization; broncho-pneumonia, affecting several points; acute œdema of the lungs, with suffocative dyspnœa. The *digestive* forms may be choleraic with profuse serous diarrhœa, cyanosis, and shrinking of the tissues, or dysenteric with bloody stools. The *renal* forms have acute nephritis. In the *cardiac* forms occur collapse, irregular pulse, dyspnœa,

cyanosis, and paroxysmal tachycardia. The *nervous* forms may be shown by convulsive symptoms, or motor paralysis due to meningo-encephalitis.

In some areas, regular epidemics of streptococcic empyemata have been reported.

In all these forms, the prognosis is always grave. There is often a remission of the pyrexial and general symptoms on the second or third day, but the temperature soon runs up again to 105°, dyspnœa become extreme, and death follows quickly.

Treatment.—In 1888, Ch. Fiessinger prescribed on the first day a purgative or an emetic, and on the following day a draught containing 70 to 80 cg. of sulphate of quinine, together with, in the case of diarrhœa, subnitrate of bismuth. In the respiratory forms, he made use of red sulphurated antimony and ergot, allowing the patients milk, broth, and *thé au rhum*.

In the present epidemic, the following measures are recommended. As a preventive, contact with a sufferer should be avoided, and such patients should be isolated. Cases of pneumonia should be kept apart from those of broncho-pneumonia. The medical man in attendance should wear an overall and a gauze respirator. This gargle should be used every three hours :—

Phenol	10 g.
Alcohol	10 g.
Water	100 g.

" One teaspoonful in a claretglassful of warm water for gargling."

Instead of this may be used a few drops of the following in half a glassful of water :—

Thymol	3 g.
Tincture of vanilla	10 g.
Alcohol (80°)	115 g.

Night and morning, a few drops of 10 per cent. solution of gomenol in oil should be instilled into each nostril.

Before the two chief meals of the day, it is well to take a cachet containing 0·25 g. of sulphate of quinine.

In an actual attack, if mild in character, the gargling and instillations should be continued, and either from 0·50 to 0·80 g. of quinine or from 0·50 g. to 1·0 g. of hexamine should be taken each day. Every three hours, a tablespoonful of the following is advisable :—

Ammonium acetate	4 g.
Sodium benzoate	4 g.
Compound tincture of camphor	5 g.
Simple syrup	150 g.

For intense headache, small doses of an analgesic—0·15 pyramidon, 0·25 acetyl-salicylic acid—may be given. Large doses should never be used foi fear of collapse and serious cardiac symptoms.

Diarrhœa should be treated with collargol, thus :—

Collargol	0·20 g.
Distilled water	180 g.

" One tablespoonful every four hours."

For dyspnœa, pneumonia, or broncho-pneumonia, hot fomentations covered with oiled silk should be applied to the chest every two hours, and

at night dry-cupping in 20 places over the back.

The wet pack is necessary every four hours for temperatures above 104°, the patient being kept in it for 20 minutes, or a bath (82° to 93°) for 10 minutes to a quarter of an hour every three hours.

In urgent dyspnœa, Ravout advises bleeding to about 500 cc. In cardiac symptoms, hypodermic injections of sparteine (2·5 to 5 g. in the day) or of camphor and ether in oil are preferable to caffeine.

During the febrile stage, fluid diet containing as much sugar as possible should be given freely in the form of lemonade, coffee and milk, plain water, or diluted milk. After the temperature has fallen, ordinary diet should be resumed gradually. The extreme weakness that follows an attack will be improved by giving before each meal a tablespoonful of the following :—

Strychnine sulphate	0·04 g
Sodium arseniate	0·02 g
Quinine wine	300 g.

Leyroux recommends as additional treatment the use of a mixture of 20 cc. antistreptococcic serum, 20 cc. of antipneumococcic, and of a serum prepared during the present epidemic against Pfeiffer's bacillus.

ANTIPYRETICS IN INFLUENZA.

Fever being a defensive reaction, it is hazardous to attack the fever except through the cause. The danger is all the greater in influenza, for it is a disease with a tendency to produce collapse. Mild cases recover with little or no treatment, but severe cases are another matter. Collapse has occurred on the fifth day of the disease in patients who have only taken three or four cachets containing 0·50 g. of aspirin to reduce the temperature. The reduction has been obtained, but the patient has died. Aspirin is an excellent remedy, specific for subacute rheumatism, and most effective for migraine and other painful symptoms; but in pyrexial conditions not more than from 0·25 g. to ·50 g. should be given, if the patient is in great pain in the early stage. None should be allowed afterwards.

For antipyretic treatment, there are only three methods which are free from harmful effects, because they attack the cause and not the effect. These are hydrotherapy, collargol, and metallic ferments.

For a temperature of 104° to 105°, a wet-pack should be applied to the chest every three hours, covered with cotton wool and protective, or warm baths (86° to 96°) given for 10 minutes every three hours. These will ensure the combustion and elimination of the toxins, and improve the general condition. Netter is content with the inunction of 15 per cent. collargol ointment once a day. A. Robin uses intra-muscular injections of 10 cc. of metallic ferments from the third day, repeating it each day. Seven hours after the injection he prescribes a cachet containing 0·30 g. of pyramidon and 0·60 g. of quinine hydrochloride.—(*Journ. des Praticiens*, October, 1918)

THE HISTORY OF INFLUENZA.

THE latest bacteriological investigations on this protean disease leave us with no definite information with regard to the actual specific organism concerned. It must accordingly be relegated to the class of infectious diseases comprising, oddly enough, those most commonly met with, to which Ovid's phrase, *causa latet, vis est notissima*, can be applied much more exactly than to the original fountain in the fable he was relating.

As Creighton points out, it is very difficult in the early records to differentiate between ague and catarrh, with both of which affections influenza is closely allied in the more prominent symptoms. It is, therefore, almost a matter of certainty that the disease has existed from the earliest times, and that those medical historians who believe that the epidemic mentioned by Hippocrates and Livy as occurring in 412 B.C. was influenza are right, as well as those who connect passages in Thucydides with the disease.

There were epidemics from the sixth to the tenth century which certainly relate to influenza, being characterized by catarrhal symptoms and cough, but Hirsch considered that they were not characteristic enough to include in his survey, and accordingly began with December, 1173, as the first authentic epidemic; this occurred in Italy, Germany and England. Other writers take later dates, such as 1239 (Zerviani), 1323 (Gluge), 1387 (Schweich and others), and 1510 (T. Thompson). In this country it was prevalent in 1173, as "a certain evil and unheard-of cough" affecting everyone and proving fatal to many.

In the record kept at St. Albans, 1423–1431, the unknown chronicler makes an entry in 1427 to the effect that "In the beginning of October a certain rheumy infirmity, which is called *mure*, invaded the whole people, and so infected the aged along with the younger that it conducted a great number to the grave." Creighton considers this to be undoubtedly influenza. In Paris, during the same year, a similar disease was prevalent generally, and was known as *ladendo*; this, too, from the records is considered to be undoubtedly influenza.

The first pandemic of the disease occurred in 1510, when it was generally diffused throughout Europe, though no mention is made of its appearance in this country except in a foreign account. Sweating is noted as one of the symptoms, and Creighton suggests that the ill-health of Erasmus—attributed by him in a letter from Queens' College, Cambridge, in August, 1511, to a sweat—was very likely due to the after-effects of an attack of influenza in the previous year. The disease spread northwards from the shores of the Mediterranean, and

was called *cocqueluche* in France and *coccolucio* in Italy.

Hirsch, in his chronological survey of epidemics occurring between 1173 and 1875, tabulates no less than 299; but this number includes small local epidemics as well as the widespread pandemics. Leichtenstern gives the following list of the most noteworthy outbreaks of the disease.

1510.—Wide distribution over Europe. Direction in general from south to north. Malta, Sicily, Spain, Portugal France, Germany, Holland, England, and Hungary.

1557.—Asia, Constantinople, Sicily, then towards the north. Italy, Switzerland, France, Spain, Holland.

This country was also included in the area affected, for Creighton quotes a contemporary writer, Wriothesley, who states that, in 1557, "This summer reigned in England divers strange and new sicknesses, taking men and women in their heads; as strange agues and fevers, whereof many died." Stowe records in his *Annales* that, in the harvest of 1558, "quartan agues continued in like manner, or more vehemently than they had done the last year passed, where-through died many old people and specially priests, so that a great number of parishes were unserved and no curates to be gotten, and much corn was lost in the fields for lack of workmen and labourers."

1580.—First real pandemic; at first, a general distribution over the Orient, then Constantinople and North Africa. In Europe the general direction was from east to west and from south to north. From Constantinople to Venice, Sicily, Italy, France, Spain, Portugal, Hungary, Bohemia, Germany, Holland, Belgium, England, Denmark, Sweden, and Livonia.

Creighton points out that these widespread volatile sicknesses of 1557–8 and 1580–2, grouped under influenza, were related in time to great epidemics of bubonic plague. These, however, were less noticeable in England than abroad.

Seventeenth Century.—Very scanty reports concerning influenza. In 1627, a great epidemic occurred in North America, spreading thence to the West Indies and South America as far as Chile.

Hirsch records an outbreak in the New England States in 1655. Creighton calls attention to the account written by Willis of three consecutive epidemics in the autumn of 1657, the spring of 1658, and the autumn of 1658, the middle epidemic of the three being influenza, as being the first systematic piece of epidemiology written in England. The doctrine of an epidemic constitution of the season, and that of a seasonal predisposition in the human constitution to which, later, Sydenham devoted many essays, were generally held in those times.

The "Father of English Medicine" gives a special chapter in his *Observationes Medicæ* to the "Epidemic Coughs of the year 1675, with Pleurisies and Pneumonia supervening." The epidemic spared hardly anyone of whatever age or temperament; it went through whole families at once.

Evelyn, in his diary, states on October 15, 1675: "I got an extreme cold, such as was afterwards so epidemical as not only to afflict us in this island but was rife all over Europe like a plague."

An epidemic occurred in London and in Ireland in 1688 in which very many people suffered, but very few indeed died from the disease. In the account given by Molyneux of the disease in Ireland he mentions that nearly all the horses suffered too. At the Curragh, where the army of Ireland

was encamped, not ten horses in a regiment escaped.

1709–12.—Probably a single period of epidemics of wide distribution but no particular direction. The mortality was very low.

1729–33.—First invasion beginning in 1729, with the direction from east to west: Russia, Sweden, Poland, Germany, Austria, Hungary, England, Switzerland, France, Italy, Iceland, and possibly America.

Second invasion in 1732, probably beginning in Russia. The direction then went definitely to Poland, Germany, Switzerland, France, England Italy, Spain, and America. Hirsch states that it was apparently a genera diffusion over the globe.

1743.—Spread from the coasts of the Baltic to Germany, Switzerland, Italy, France, Netherlands, and England.

1757–8, 1761–2, and 1767.—Epidemics first arose in North America and thence appeared to spread to Europe. Widely diffused in America and West Indies.

Heberden read a paper in August, 1767, at the Royal College of Physicians on "The Epidemical Cold in June and July, 1767."

1781–2.—Decided pandemic, beginning in the autumn of 1781 in China and India, and from thence to Siberia and Russia; then generally over the Eastern Hemisphere.

The Italian name for the disease was formally adopted by the College of Physicians. The name first appeared in English in 1729 in an account of an Italian epidemic. In 1743 the name reached London before the infection, and the disease was so called during the epidemic of that year. The name came into general use in the epidemic of 1775. Creighton remarks that the adoption of this name at last put an end to the ambiguity between epidemic agues and influenzas.

1788–90.—A most pronounced pandemic period. It began in Russia and spread thence over the rest of Europe. In 1789 it became generally diffused throughout America.

1799–1803.—A long period consisting of several invasions and outbreaks of the disease. The first pandemic began in Russia in 1799, and spread to Galicia, Poland, Germany, France, and Denmark. After an interval of five months, a second began in October, 1800. In 1802–3 it was extensively diffused in France and in Britain, with minor outbreaks up to 1808. Between 1811 and 1826, there were numerous epidemics in North and South America.

1827.—Generally diffused through Siberia and Eastern Russia.

1830–3.—A remarkably intense influenza period, distributed over the whole world, and consisting of two or three pandemic outbreaks. The first invasion began in China (1830–1), and spread to Manila, Polynesia, Borneo, Java, Sumatra, and India. The European invasion began in Russia in October, 1830, and advanced in 1831 through Courland and Livonia, Poland, East Prussia, Silesia and the rest of Germany, Austria, Finland, Denmark, Belgium, France, Sweden, England, Scotland, Switzerland, Italy. It reached Spain in January, 1832, and North America at the same time.

A year later a fresh outbreak occurred in Europe, which was of an exceptionally intense character, and travelled from east to west.

A third outbreak originated in Russia in 1833, and was practically identical

with that two years before in its course. Three years later a new pandemic invasion travelled right round the world.

1836-7.—After raging in Australia, South Africa, Java, and Further India, influenza spread from east to west in Europe, beginning in Russia and extending rapidly. A primary northern outbreak can be distinguished, spreading from east to west, and a subsidiary outbreak from this point, with the direction from north to south.

1847-8.—The direction of this pandemic cannot be determined very definitely. An epidemic occurred in 1846–7 in England, Denmark, Belgium, Switzerland, and France, and this was followed in March, 1847, by an outbreak in Russia. The principal outbreak occurred in September, 1847, appearing first in France, and from thence extending quickly to Germany, Denmark, England, Scotland, Switzerland, Italy, Spain, and Greece. North America was affected in January, 1848.

1889-90.—The most extensive and important of all the pandemic outbreaks, which extended all over the world. It was first observed as an extremely intense outbreak in the interior of Turkestan in May–June, 1889. It took four months to spread to Eastern Russia and Siberia, and five months to reach Petrograd.

From Russia and Finland in November it spread like an avalanche over the whole of Europe. It was general in Germany and Switzerland by the beginning of December, but did not reach London until the second week of December. By the end of the year the whole of Europe was affected. The disease appeared in New York and Boston in the middle of December, and from thence spread over the Continent, appearing in Quebec, Montreal, and Halifax in January. The north and south of Africa were affected simultaneously. It was established in China by the end of February, by which time, too, it had spread to Australia, New Zealand and Borneo.

By degrees all places on the habitable globe were infected, and the last appearances were in Abyssinia in November, and in the highlands of Kashmir in December, 1890. This pandemic spread with far greater rapidity than any of its predecessors. In 1830–1, after first appearing in Moscow, it was eleven months in reaching Spain and North America. In 1889–90, in the same period of time, the infection had covered the whole of the world and extended up into the heights of the Hindu Kush range of mountains.

The disease has had bestowed upon it a plethora of names, both medical and popular. In the early years, it was variously described as *catarrhus epidemicus, febris* or *cephalea catarrhalis epidemica, tussis epidemica, contagious catarrhal fever.* The severity or mildness of its epidemic manifestation can be gauged by the character of the name, the droller names pointing to the milder forms.

In the fifteenth century, the English name was "mure" or "murre," the origin of which is allied to murrain. In the sixteenth century, it was known at different times as "hot ague," then "the new burning ague." This was followed by various cant names such as "the new acquaintance" and "the gentle correction." In the seventeenth century it was known variously as "the new disease," "the new ague," "the strange fever," "the new delight," and "the jolly rant." The names "tac," "sheep's cough," and "sheep's disease" were obviously bestowed on account of the loud bleating

cough accompanying it. Robert Boyle spoke of one sudden outbreak as "a great cold." Horace Walpole called it "blue plagues."

In Ireland, in a medical manuscript of the fifteenth century, it is called *Fuachd* (cold or chilliness) and *Slaodan* (a cough or cold). It is alluded to in some early Gaelic manuscripts under the name of *Creathan* (a shaking or trembling).

In France, the name of *la grippe* first came into use in 1712, at which time it was called in Germany *Galanteriekrankheit* and *Modekrankheit*. These names bear witness to the prevalence of the disease at the time, and that it was the fashionable disease from which to suffer.

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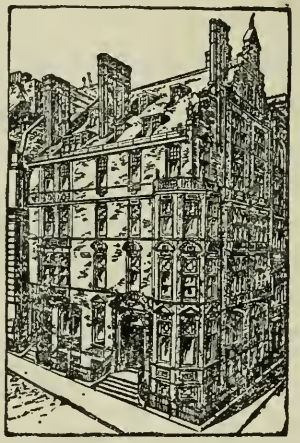

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